UNITED STATES BANKRUPTCY COURT
FOR THE WESTERN DISTRICT OF NORTH CAROLINA
CHARLOTTE DIVISION

IN RE:

GARLOCK SEALING TECHNOLOGIES, LLC, et al,
Debtors.

No. 10-BK-31607

VOLUME VII-B
AFTERNOON SESSION
TUESDAY, JULY 30, 2013

TRANSCRIPT OF ESTIMATION TRIAL
BEFORE THE HONORABLE GEORGE R. HODGES,
UNITED STATES BANKRUPTCY JUDGE

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I N D E X

	DIRECT	CROSS	REDIR	RECROSS
Arnold Brody	1847	1867	1908	1914
Carl Brodkin	1916			

E X H I B I T S

Debtors' Exhibits No.:	ADMITTED

ACC's Exhibits No:	ADMITTED
ACC-3562	1866
ACC-3563	1866
ACC-3564	1866
ACC-3565	1866
ACC-3332	1990
ACC-3333	1990
ACC-3334	1990
ACC-3336	1990

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P R O C E E D I N G S

(On the record at 1:47 p.m.)

MR. GEORGE: Good afternoon, Your Honor.

THE COURT: Good afternoon.

DIRECT EXAMINATION CONTINUES

By MR. GEORGE:

Q. When we left, Dr. Brody, we talked about the fact that this is a diagram of the pleura and how the fibers get to the pleura. Do you have a diagram that shows where Mesotheliomas occur and what they look like when they do?

A. Yes. You can see, again, on this diagram. This is the lung. And normally, as we saw earlier, the pleura should have a -- should be very thin. I mean it's normally Saran Wrap thin, that's how thin it is, and then with a single cell layer on the outside. And then when there's a Mesothelioma present, there's a dramatic thickening as the tumor cells build up on either side of the lung, the so-called visceral pleura or the parietal pleura under the ribs. The tumor can grow from either side and grow into the peritoneal cavity as well and into the structure of the lung.

Q. Now, do you have some slides that will show us whether the asbestos fibers have the ability to cause that type of cancer?

A. Yes. So, in order for asbestos or any carcinogen that's a cancer-causing agent to cause a cancer, it has to cause what's called "genetic damage." It has to damage genes. So this is the cover of a proceedings -- of a meeting I was at a few years ago, and the topic was how fibers cause cancer, carcinogenesis cancer formation. I gave a talk at this conference. I've talked to you today about cells and I've showed you that cells can pick up fibers, but you can't talk about carcinogenesis unless you talk about the molecular aspects. That means your genes, because cancer is a genetic disease.

The simplest definition of cancer is the loss of control of cell growth. Cancer is the loss of control of cell growth. Humans have about 20,000 or so genes that make us what we are. Of those 20,000 or so genes, about 100 of them control cell growth called growth control genes. Some of them, in fact, are dedicated to protecting us against cancer, from getting cancer, and they're called tumor suppressor genes, for example.

In order for a carcinogen to produce a cancer it has to cause errors in a series of genes that control cell growth. A series of those genes -- I told you those hundred or so genes that control cell growth. So one of the ways that scientists can establish how asbestos or other carcinogens cause that change in cell growth and

genetic damage is by taking cells out of animals or out of people, putting those cells in a dish, a so-called in vitro study. If you add the right nutrients to those cells, they'll continue to grow. And you can actually, then, add the carcinogens and study the interactions between the carcinogens and the DNA.

Q. This can be done with both animal cells and human cells?

A. Yes. It's done regularly like that. That's right. So, for example, on the cover of these proceedings there were two cells. I'm outlining one of them for you here, and then there's another cell over here. And some fibers have been added, and you can see there's a long fiber there and some short fibers, and those fibers have collected around the center circle in the cell. And the center circle is called the nucleus. And the nucleus of our cells contains all of our DNA.

I told you that "molecular aspects" means your genes. And when we're talking about our genes, we're talking about DNA. Our genes are made up of DNA, short segments of DNA. Now notice, how the fibers have been excluded from the center circle. And they're excluded because there is a membrane that surrounds the nucleus, nuclear membrane that protects all the genetic material. That's why it's excluded. That's a good thing. That's

what we expect to happen.

Now, it turns out that when cells are dividing, when we're making new cells, that nuclear membrane dissipates and the nuclear membrane is no longer there to protect the DNA. So we asked in my laboratory, what would happen if we added fibers when the cells were dividing? And I can -- I can show you what happens.

Q. Now is that cell division process a process that's ongoing throughout the time that we live?

A. Right. From the time after the first division of the egg in the womb. I mean that's -- that's the same similar kind of cell division. And every time there's --

Q. When we're standing here, are some of our cells dividing?

A. Exactly. Some of our cells are dividing. And what you'd expect them to do is what I'm exactly showing you here. Here are three cells: One, two, and then three. There are three cells. The two cells on the outside are not dividing. You can see there the DNA's been stained blue and it's contained in the nucleus. This cell in the center has received a signal to divide. It could be just normal growth rate. For example, your skin. About ten percent of your skin cells are growing replacing themselves. One percent of your lung cells. The mesothelial surface has a very low rate of one-half

of one percent, but eventually they all have to be replaced.

Now this cell in the center has received a signal to divide. Perhaps its neighbor was injured. I could have added a growth hormone. Whatever the reason, this cell's divided. Now that means what's happening is that the DNA in the nucleus has condensed into these white threads called chromosomes. And what we're trying to do then is make a perfect copy of all of the DNA. So what has to happen is that the chromosomes are going to duplicate and then we'll get two new cells like the original. And let me show you what your chromosomes look like.

Humans have 23 pairs of chromosomes. Each chromosome, one -- you've got one from your mother and one from your father. And lined up on the chromosomes are these light and dark bands that represent where our 20,000 or so genes are distributed. Now, the point of this is that each gene must be in the correct place on the correct chromosome. There's no mixing and matching of where our genes can be located. In order to function correctly, the gene must be in the correct place on the correct chromosome. So if we finish this process of normal cell division, the chromosomes have condensed. They replicate. And if they go through faithful

replication you'll get two what are called "daughter cells." That's what we hope for every time.

Now, this is an experiment using cells. You can see here a normal cell. This is one of millions of cells in the experiment, and there's -- there are no fibers. On this side, in panel B, crocidolite fibers have been added and you can see there's long crocidolite fibers and some shorter ones. In this slide with no fibers, half the chromosomes moved to one side and half to the other. We'll get two new daughter cells.

On this side, most of the DNA has moved to the new developing cells but some of the DNA is bound to the surface of the fibers resulting in this condition called Aneuploidy. Aneuploidy means abnormal chromosome separation. And let me show you that Chrysotile does the same thing here. Here is a normal mesothelial cell. The mesothelial cell here has no fibers. So half of the chromosomes will go to one side, half to the other. We'll get two new daughter cells. Here you can see the two daughter cells have formed and there's a Chrysotile fiber spanning the two cells and there's DNA bound to the surface of the fiber, again, producing Aneuploidy.

Q. Now the way those Chrysotile fibers get in, because during the process of separating we lose that barrier?

A. That's right. The barrier that protects the DNA nucleus is lost during cell division. And it's been known for a long time that dividing cells are more likely to become cancer cells because of that reason. They don't have that protective envelope.

Q. If this happens once, are you going to get cancer?

A. No.

Q. What happens if it happens twice?

A. No.

Q. What does it take for a cancer to form?

A. Okay. So, that is actually answered in the last slide. But let me first tell you the significance of this DNA bound to the surface of the fiber, if I can.

Q. Sure.

A. So, I told you a minute ago when I showed you the chromosomes that every one of our genes must be in the right place at the right time in order to function. So let's take, for an example, a gene that we know very well that I've studied that's called P-53. This is a tumor suppressor gene. When a cell gets DNA damage, the P-53 gene is activated and stops the cell from dividing. If the cell is not dividing it can't pass on mistakes or genetic damage to the daughter cells. So that's how it protects us.

We have another set of genes called death pathway

genes. When there is DNA damage, these death pathway genes get activated and drive the cells down our death pathway. The cells die and you never hear about them again. And this is going on in us all the time when we're exposed to carcinogens in the environment, whether it's ultraviolet light from the sun or cigarette smoke. These genes get activated and protect us from getting cancer. If in this DNA that's bound to the surface of the fiber is a gene that we need to protect us, it's not going to work.

Q. So those suppressor genes are much like the body's defense mechanisms for the fibers getting in the cilia and all that. These are molecular defense mechanisms against foreign and particulate matter?

A. These are molecular defense mechanisms that protect our Genome, our genes, from carcinogens, from damage from carcinogens. That's right.

Now, this is the end of the cancer description slides that I have because there's a lot going on in this time period that we call latency. So, you know, from the time for first exposure until the time the person comes to the clinic. So it's good to understand what's going on during those decades related to what I just told you.

Q. Okay.

A. So if you take this mesothelial surface and see

the individual cells with the single nucleus. And here you can see the artist has given us a couple of lightning bolts and he says "DNA damage" from something from the environment is what he's meaning, something that's coming in from the outside and reaching the DNA. Now, in this case it could be asbestos. We're talking about Mesothelioma. Obviously, it's asbestos that can either bind the DNA as I just showed you, but asbestos can also generate what are called "oxygen radicals." These are short-lived, high energy compounds that are known to cause DNA damage.

So, asbestos has sort of a double whammy in its ability to produce genetic errors. Number one, it binds DNA. Number two, it binds oxygen radicals. So whatever the specific damage, this is a general discussion of DNA damage. And what the artist has done, he knows very well that typically when there's DNA damage the cells die. So he has one cell going up into here to the left-hand corner and dying. The DNA's all clumped up. The surface of the cell is bubbling up and the cell is going to die and you never hear about it again. But we're talking, obviously, about a cancer. We know the cancer has developed. So that means one of the daughter cells with a genetic error must have survived. So, here is that daughter cell.

Then the artist has a tumor growing out here and he calls it "tumor genesis" or tumor formation. And you can see there are multiple tumor cells with odd combinations of DNA. And this distance, this time between the first daughter cell and the generation of the tumor, is that latency. So you've got to give me about 20 years there, 40 years there. And I'll just take a few seconds to explain what's going on in that latency time.

So, think about this one mesothelial cell sitting on the mesothelial surface among the hundreds of millions of mesothelial cells we have and the cell's looking and acting just like a normal mesothelial cell. There's no way you'd know it was there unless you went in and sequenced the DNA on every mesothelial cell on that person's surface and, of course, that's not going to happen. So that cell then can sit there like that with that error in a gene that controls cell growth. It can sit there like that for months, but eventually it has to divide. Two cells, four cells, eight cells, 16 cells passing on that error to the other cells, to the new cells.

Now, one or more of them might die. But then in order to get a cancer you have to have a second error. Another asbestos fiber comes in and hits one or more of those new cells. Now it has two errors. And that cell,

then, with the two genetic errors divides and can sit there looking like -- looking and acting like a normal mesothelial cell for months. But eventually it has to divide: Two cells, four cells, eight cells, 16 cells. Some of them may die. One or more of them gets hit again. Three errors go through the scenario again. Four errors.

Q. When you say "error," what you mean is you have the daughter cell's problems. And then when it gets hit again it creates another rearrangement, so that creates a different replication.

A. That's right. Another gene. Because we haven't told you about a hundred cells that control cell growth. What we're talking about here is an additional gene that controls cell growth now is damaged.

Okay. Now you go through that scenario of cumulating errors for decades. And then eventually, at the end of the time that it takes to accumulate sufficient errors for that person, because you see how the artist has given us this oddly colored tumor? It's oddly colored because that tumor -- all the cells in that tumor came from a single cell with a sufficient combination and number of errors for that person. Now we know how many errors you can find in a given tumor, but it's going to be different with a different combination

among different people.
Q. Is that what you call individual susceptibility?
A. That's part of the individual susceptibility and it's part of the individual's response, of course. But eventually the tumor grows out and, of course, that's what brings this person to the clinic.
Q. Now how does the multiple errors, how does that impact on the dose-response and the cumulative nature of asbestos in causing this?
A. Sure. The more a person's exposed, the more likely it is that a fiber or series of fibers is going to be carried to the pleura and be able to interact and produce the genetic errors that I just described.
Q. And then you have a final slide on summing up your testimony with regard to Chrysotile?
A. Yes.
Q. So from what your studies show, does it show that Chrysotile asbestos is highly toxic to both human and animal mesothelial cells?
A. Yes. I've done some of this. But there are a number of studies -- I've not studied human mesothelial cells in culture but others have and, clearly, it is toxic.
Q. All right. And in your studies, have you -- and in your research have you seen the fact that Chrysotile

can cause scarring inside the lungs of rats, mice and humans?

A. I've published a number of papers showing that. Yes.

Q. And that scarring inside the lung tissue is called what when it's caused by asbestos?

A. Asbestosis.

Q. So can Chrysotile leave the mineral fiber itself and cause asbestosis in humans and in animals?

A. No question.

Q. Okay. Is Chrysotile cytotoxic to the human macrophages?

A. Absolutely. And I've studied that in my laboratory as well.

Q. The macrophages are like the scrubbing bubbles in lungs and our cells. They go along and try to eat up toxic --

A. They try.

Q. What happens if something is cytotoxic?

A. Cytotoxic means all the macrophages, the varieties are toxic itself.

Q. And if you kill off your macrophages, what effect does that have on your body's ability to handle the asbestos that you inhale?

A. Well there are a couple of things going on there.

First, the macrophages, if they are toxic and cannot actually clear the fibers out of the lung very well. And then also if there is a response of these macrophages, that plays a role in the development of the scar tissue disease.

Q. Okay. Now, are -- is Chrysotile fibers, are they mutagenic to the cells and do they damage DNA?

A. Yes.

Q. What does "mutagenic" mean?

A. So, mutagenic means that you're causing errors in specific genes. The concept of asbestos, and particularly Chrysotile being mutagenic, has been shown by several different investigators. The work that I did was more related to a DNA damage, not to mutagenesis. But other scientists have shown that.

Q. And have your studies and others shown that Chrysotile causes Mesothelioma, not only in rats and mice but also in humans?

A. Yes.

Q. Let me just ask you one thing before I ask you to take your seat. There have been many different researchers that have done inhalation studies with different types of animals; correct?

A. Yes.

Q. Is the inhalation model a good model to look for

Mesothelioma causation in animals? Do all animals get Mesothelioma from inhalation of asbestos?

A. No. I don't think that's really a good model to study causation. The animal models are best for understanding how asbestos causes the diseases in the species. We know it does, like, in people and rats and certain animals. Yeah.

Q. So in order to induce Mesothelioma, what do researchers do besides the type of aerosol asbestos experiments that you do? Do they do injection experiments?

A. Yes. Sure. So you can inject the fibers directly into the peritoneal cavity, for example, or into the pleural cavity. And that's a very effective way, with all the asbestos ways, of producing Mesotheliomas.

Q. The researchers that do this, are they doing that to establish causation, or are they doing that to establish pathways of carcinogenesis?

A. Yes. In the latter, I think, early on in the years of animal experimentation, the idea was to ask if those kinds of studies could tell you about causes. But they're much better at -- those animals experiments are much better at understanding how the agent causes the disease.

Q. Now I know you've been asked this in other trials.

Has medical science determined how Mesothelioma develops?
A. Well, it really depends on the level at which you're asking the question. I mean, if you ask me do I know the precise genes that it takes for a given individual to create a Mesothelioma? No. You can't do that. But if you ask me do we know that it requires a series of genetic errors caused by oxygen radical production or by DNA binding? Well, sure, we know a lot about those mechanisms.
Q. And from your research and what you've viewed in the peer reviewed medical and scientific literature, is there a potency difference between the different types of asbestos?
A. There does -- there seems to be. Yes.
Q. What's the hierarchy?
A. Well it looks like crocidolite is probably the most potent and Amosite next and then Chrysotile, but I don't really know that there's much difference. I don't know the basis for actually determining a difference between crocidolite and Amosite, but there does appear to be some difference and that's variable. I really don't have a number for the real difference.
Q. Has anybody been able to scientifically establish the precise potency difference from one fiber to the next?

A. I would say no. And the reason I say that is because if you look in the literature, there's a huge range. I've seen everything from two times more potent to hundreds of times more potent. And some investigators who tried to nail that down have changed their numbers.

Q. And to the extent that there is a potency difference, we're talking on a fiber-per-fiber basis; correct?

A. That's right.

Q. So if you have more quantity of a less potent fiber, how does that compare to less quantity of a more potent fiber?

A. Well that's what that means on a fiber-by-fiber basis. So if somebody says, well, this kind of asbestos is a hundred times more potent than Chrysotile, let's say. So that means you need a hundred Chrysotile fibers for every single crocidolite fiber or Amosite fiber, and then you have equal potency. Okay. Well, what if you have a mineral where you have billions or millions of Chrysotile fibers for every one Amphibole? The issue of potency wouldn't make much difference there I wouldn't think.

Q. Does the fact that the Amphiboles are more potent negate the potency, the capability of the Chrysotile fibers to cause disease?

A. Not at all.
Q. So if you have a person that's exposed to a majority of Chrysotile and a minority of some sort of Amphibole, can we discount all of the Chrysotile exposure and say it must have been the more potent fiber that caused the disease?
A. I don't know how you can do that.
Q. You've read many articles about asbestos and Mesothelioma. How many articles do you think have been written about asbestos and Mesothelioma as a cause from asbestos exposure?
A. Oh, there must be, I don't know, hundreds. Thousands. I'm not sure.
Q. Has anybody been able to determine to a reasonable degree of medical certainty or scientific certainty what level of exposure somebody could have to asbestos that would prevent them from getting Mesothelioma?
A. No, not that I know. And it's so different for different people, I don't know how one could do that.
Q. You also are aware, I think, and there has been testimony in this case -- there are people that have not been occupationally exposed to asbestos who, when they do an autopsy on them, they've found millions of asbestos fibers in their lungs. Have you seen it?
A. I've seen it and I've published papers like that.

Sure.
Q. What is the significance, numerically, of having millions of asbestos fibers in your lungs?
A. Sure. I mean there's a huge range of what can be found in people's lungs. It depends on where you lived and whether or not you worked with asbestos. So if you have millions of fibers that, in and of itself, doesn't say you have had much of an exposure because, I mean, you can fit a billion fibers into a thimble. So, I mean, that doesn't sound like a lot.
Q. The last thing I wanted to cover with you is, there's been some testimony that epidemiological studies have shown that anywhere from 80 to 90 percent of Mesotheliomas are caused by asbestos exposure, meaning there's ten to 20 percent that they call idiopathic. You've read literature like that?
A. Yes.
Q. Does the fact that the cause of a tumor is determined to be idiopathic mean that it's not an asbestos-related tumor?
A. No. It doesn't mean that. It just means you don't know the cause. You haven't recorded a cause.
Q. And what are -- what are some of the factors in the development of Mesothelioma that may prevent medical scientists and doctors from determining a cause of a

particular Mesothelioma in a patient?
A. They might not have a clear picture of that person's history. That would be the most likely possibility for me.
Q. What's the average life expectancy of somebody once they've been diagnosed with Mesothelioma?
A. It's not much. About 18 months is about the average.
Q. Would that have an impact on the ability of researchers looking backwards to try and ask that individual questions about his exposure?
A. Well, of course.
Q. And then you recognize there's a latency period between when the exposure first occurs and when the disease develops.
A. Right.
Q. What impact does the fact that there may be 30 or 40 years from that initial exposure have on the ability to determine whether that individual was exposed to asbestos or not?
A. I'm sure people can forget things.
Q. Your Honor, at this time I would like to offer the Curriculum Vitae which is ACC-3562. His initial report which is ACC-3563; the supplemental report which is ACC-3564. And for identification purposes, I've printed

out a copy of the slides that I've shown, and that's marked as ACC-3566.

MR. SCHACHTER: No objection, Your Honor. Sorry.

MR. GEORGE: Offer that.

THE COURT: Okay. It will be accepted.

THE COURT: Okay. Mr. Guy. Mr. Schachter, Mr. Guy is going to go next.

CROSS-EXAMINATION

BY MR. GUY:

Q. Dr. Brody, my name is Jonathan Guy. I represent the future claimants representative in the case, Mr. Grier, who is here in the courtroom. Happily, in this case we're not trying to reach a definitive ruling as to whether Chrysotile asbestos causes Mesothelioma. We're just trying to determine whether there's a credible debate on either side of that issue. I want to ask you about how long that debate has been known in academic circles. If we could pull back up the last slide that had the various --

MR. GEORGE: Sure.

BY MR. GUY:

Q. You testified as to those issues concerning Chrysotile. Have you testified to those issues before in court?

A. Sure.

Q. Many times?
A. Yes.
Q. How long have you held your opinion concerning those issues?
A. Well, I started my work with Dr. Chris Wagner in 1974. So that's been my understanding since then.
Q. And you've published on these issues; correct?
A. A number of times. Yes.
Q. And have you ever testified in a case where Garlock was the defendant?
A. I'm sure I have.
Q. Do you have any reason to believe that Garlock would be aware of your opinions concerning these issues in the 2005 to 2010 timeframe?
A. I don't know why they wouldn't be. No.
Q. Now, are you aware of a medical doctor at Stanford University, Dr. Weill?
A. Yes.
Q. He testified earlier. Were you here to hear his testimony?
A. No.
Q. Are you familiar with whether he has opinions concerning whether Chrysotile asbestos causes Mesothelioma?
A. Some of them. Yes.

Q. He testified, I believe, and I'm paraphrasing here because I don't have it verbatim. But he said, I think, on the stand, fairly candidly, that there is debate in academic circles as to this issue. Would you agree with that statement?
A. No.
Q. Why would you disagree?
A. Well, "academic circles" means to me multiple places where these kinds of discussions would be debated. I'm in academic circles all the time and I don't hear those debates. Occasionally, if there's a large meeting dealing with asbestos, somebody might bring it up or present a paper. But there's not much of a big debate other than in the courtroom that I know about.
Q. But there is a debate; correct?
A. There is a debate. Sure.
Q. Thank you, Your Honor.
THE COURT: Thank you.
All right, Mr. Schachter.

CROSS-EXAMINATION

BY MR. SCHACHTER:
Q. Good afternoon, Dr. Brody.
A. Good afternoon.
Q. Is this working? Can you hear me?
A. Yes. Fine.

Q. I must have a head cold. I hate to take issue at the start with my learned colleague, but in this case we're dealing with gaskets and packing and not with Chrysotile miners or anybody else. And the issue is about a low-dose asbestos product. Do you understand that?
A. Sure.
Q. Okay. And the issue isn't about merely whether there's a debate, but we have legal issues about methodology that applies if somebody is going to argue that low-dose Chrysotile products were a cause. So if you don't mind, I'd like to ask you questions that primarily will focus on methodology. Will that be okay, sir?
A. If I know something about the method you're asking me, that's fine.
Q. Okay.
A. And I'll let you know if I don't know anything about it.
Q. Well, you -- fundamentally, we can agree there's something called the scientific method?
A. Of course.
Q. And it starts with hypothesis?
A. Exactly. Actually, I'm sorry to interrupt you. It actually starts with an observation. And upon that

observation, then you can form a hypothesis.
Q. Thank you. That's exactly what Dr. Garabrant said. And I apologize. For some -- you need some kind of observation that leads to a hypothesis, and then you do scientific testing of the hypothesis; correct?
A. That's right.
Q. And after the tests are done you decide whether the hypothesis has been established or not established; right?
A. Whether the hypothesis has been proven or disproven. Sure.
Q. Okay. For almost all toxins -- well, for all toxins, it's a fundamental principle of science that the poison is in the dose. Correct?
A. Yes.
Q. And even for carcinogens, it is a fundamental principle that carcinogens can be dangerous at some levels but not necessarily dangerous for human beings at other levels. Is that correct?
A. True.
Q. And as you've told us, scientifically, the fact that a person may have millions or even billions of asbestos fibers in his or her lungs does not necessarily create a risk of asbestos disease.
A. Not necessarily for a given individual. That's

correct.
Q. All right. And billions can be in a thimble? We've heard a lot of what I will -- we've heard a lot of math about total fibers over years and what that may mean. That's not how scientists look at this. They look at it in fibers per cc and cumulative lifetime exposure based on fibers per cc years; correct?
A. True.
Q. And there are methodologies for determining scientifically, if we're going to use scientific methods, the levels of exposures associated with disease. And primarily, those are methods that rely on qualified certified industrial hygienists. Correct?
A. I agree.
Q. And they look at to determine exposure, and then other scientists can determine whether the cumulative lifetime dose from that source is associated with an increased incidence of disease. Would that be correct?
A. Right.
Q. Now, you have shown us some photographs. Are these photomicrographs or -- they're not that small; right?
A. No. You can call it a photomicrograph. It's an electron micrograph, however you'd like to call it.
Q. And this is taken from a lung of a rat that you

studied. Or was this from the literature, sir?
A. No. This is from one of my studies.
Q. And that rat -- you do these experiments where you put the rats into enclosed chambers; correct?
A. As I explained. Right.
Q. Yeah. And then they are given -- administered an aerosol continuously for a certain period of time; correct?
A. Right.
Q. And for a certain length of time. Sometimes you'll have -- what's the shortest period of time?
A. I've done half an hour, an hour.
Q. Half an hour, an hour, but others will be a week, a month, a year gone? As long as a year?
A. Well the longest that I've exposed animals is one day a week for eight weeks and then looked a year later. That's the longest my papers show.
Q. Okay. Do you happen to know what the duration was for this slide here?
A. Sure. This was an hour.
Q. Okay. So this is what a rat's lung looks like after one hour of an exposure to an aerosolized asbestos at a concentration of 1,000 fibers per cc; correct?
A. Correct.
Q. And you have told us that that's comparable based

on your knowledge of the literature to what insulators or miners might have experienced in an hour; right?

A. Right.

Q. You did not mean, by showing us this slide, to suggest that it is representative of what would be in the lungs of a mammal, a human being, after installing and removing a gasket; correct?

A. Correct.

Q. You have never attempted to grind up gasket material and administer that to a rat; correct?

A. True.

Q. And you agree that an exposure level at .1 fiber per cc for one hour would likely not look like that with that accumulation of asbestos on the -- in the slide. Correct?

A. I agree.

Q. And even if it were at one fiber per cc, it wouldn't look like this; right?

A. True.

Q. 20 fibers per cc wouldn't look like this?

A. That's right.

Q. Okay. Actually, in your studies, you have never yourself caused any of the rats to get Mesothelioma. Correct?

A. Well, as I was asked, we've not tried to do that.

That's a specific protocol that needs to be established in order to do that and it's been done many times; I have not.

Q. You're aware of something called the Laminar Flow that helps particulate matter navigate by the defense mechanisms in the respiratory system; correct?

A. Well, that slide you have up there is a direct result of Laminar Flow. That means that the fibers are flowing in the center of the pathway and then are intercepted by this area of the lung and that's why there's an accumulation there, which is what you get at ten fibers or one fiber per cc, just not as many fibers. But the concept is the same.

Q. But you are trying to induce disease, so you use a very pure form of asbestos that can become an aerosol; correct?

A. Right.

Q. And that form of asbestos does not have any particulate matter attached to it that might cause those fibers to tumble; correct?

A. Well you're talking apples and oranges here, I think. I mean, I don't know what you mean by "other particles." So, sure. I use just asbestos. The fibers are carried in a Laminar Flow pattern which you can actually see evidence of in this picture. So, I'm sorry.

I guess I'm not understanding where you're coming from with tumbling and other particles and things like that.

Q. Okay. We have heard from Dr. Weill, who is a -- well, he's in charge of the Advanced Lung Disease Clinic at Stanford university. And, of course, you know his father's a famous researcher in asbestos disease; right?

A. Sure.

Q. And Dr. Weill in his own right has quite impressive credentials. You're not critical of anything about his credentials, are you?

A. Of course not.

Q. Of course not. Now he has explained to us that encapsulated products have this propensity to tumble, which causes them to impact higher in the respiratory system and not reach the lower portions of the lung as readily.

A. I'm sorry. Were you finished? That may -- that may be, but that's certainly not anything I know anything about.

Q. Okay. Well, you do. Because in your deposition I asked you, "If they tumble, they can't get through that hole?" And you said, "They're more likely to be obstructed." Correct?

A. That's fine. Sure.

Q. Okay. We don't have a disagreement there. That's

all I'm trying to make --
A. That's fine.
Q. -- scientific disagreement. Now you've talked about two kinds of animal studies where asbestos is administered. One are the inhalation studies; correct?
A. Yes.
Q. And the other kind are injection studies; correct?
A. Right.
Q. And with the injection studies what the researchers do is they inject the fibers of whatever toxic material they're trying to use directly into the peritoneum, usually?
A. Usually, the peritoneum, but it's been done into the pleura cavity as well.
Q. They do that because that bypasses all the body's defense mechanisms through the respiratory system; correct?
A. Correct.
Q. And it makes it more likely they'll induce disease; correct?
A. Correct.
Q. Using that technique, ceramic fibers can cause Mesothelioma. Correct?
A. That's right.
Q. Silica can cause it?

A. Right.
Q. There are a whole host of maybe even not even things that you would call "toxins" that can be used to induce Mesothelioma in animal models; correct?
A. By injection, largely fibers.
Q. Okay.
A. That's right.
Q. And they are not, for that reason, considered causes -- Mesothelioma-causing agents in humans; correct?
A. Right.
Q. And I think you mentioned this, that the rat model really doesn't really tell us a whole lot about what induces disease in human beings. Correct?
A. Well, in an epidemiological sense, sure. But, I mean, the fact that the beginning of the question, would you agree the rat model is good for understanding the mechanisms of a disease? Well that's what it's all about. Sure.
Q. Okay. But even for this issue of Chrysotile. On the macro level, before we even get to the low-dose Chrysotile issue, you agree that these animal studies with rats are not for predicting whether Chrysotile can induce Mesothelioma in humans; correct?
A. Not for predicting but they are part of that biological plausibility because you can expose the

animals to asbestos, Chrysotile, and they get the disease. They cause the genetic errors that are required.

Q. I guess what I'm trying to focus on is whether you have a disagreement with what we have briefed to the Court as what the law is and we have -- we have briefed to the Court that studies in rats that use higher doses than occur in human beings just aren't -- aren't part of the proof of causation that it's not relevant to the quantities in the humans.

MR. GEORGE: Your Honor, I'm going to object to Dr. Brody's interpretation of what the legal standards are.

BY MR. SCHACHTER:

Q. I don't mean to ask him a legal question. I withdraw the question, sir.

You don't disagree that even scientists are very cautious to draw any kind of conclusions about doses that are unrealistic for the human beings for the product at issue.

A. Well, it depends on the question you're asking. Now are you asking, can you draw from the conclusion -- from a high dose study, can you draw conclusions about how the disease is caused in people? Sure, you can. If you're asking, can you predict whether or not a person is

going to get the disease from that dose? No, you cannot.
Q. Okay. And that's what we're about in this case. We've got groups of people that have a certain dose of disease and we're trying to figure out whether your studies are even relevant to that. And I think you've -- that's why I'm asking the questions.
Let's talk about another kind of animal study, you've talked about rat studies. You agree that there have been studies of baboons that have shown that Amosite causes Mesothelioma but the Chrysotile doesn't?
A. There is a study like that. Yes.
Q. There are monkey studies showing that Amosite causes Mesothelioma and Chrysotile doesn't.
A. I'm not sure I've seen those.
Q. You're aware of the Stettler studies?
A. I thought those were baboons.
Q. Okay. Maybe it was. May I approach the witness, Your Honor?
THE COURT: Yes.
BY MR. SCHACHTER:
Q. This was some kind of primate study. Was it baboons?
A. Yes, it was.
Q. Baboons or monkeys?
A. They're primates. That's fine.

Q. Okay. And this was published by researchers at the National Institutes of Health?
A. Right.
Q. They're certainly not part of industry; right?
A. Right.
Q. And it was published just a few years ago; right?
A. Yes.
Q. In 2008. And they did a followup of studies for a long time and they found no induction of disease with low dose exposure to Chrysotile, and they reported that that was consistent with other studies of Chrysotile exposure in animals. Correct?
A. Well, that's what that says.
Q. Thank you.
A. I think we've been through this. I looked at those studies and I have found evidence for injury in the lungs of these animals that they're talking about and --
Q. Yeah.
A. Yeah. So, yeah. You might not like that, but that's true. I looked at those animals and there is injury in the lungs of those animals. And in fact, these monkeys that Dr. Stettler went on to study had so much scars, I'm not even sure how he was able to draw any conclusions from them. But, you know, that's part of this issue where it says lack of pathologic findings with

low-dose that just isn't the case in fact.
Q. Well, actually, you looked at some of the early animals what was it 20 years ago when the first studies were published?
A. Yeah in the 1970s. Right.
Q. In the 1970s so we have 30 or how many years later after all the animals have tied and researchers at the National Institutes of Health have published in a peer reviewed journal that there's no damage. Have you published in a peer reviewed journal your views on looking at those animals from 30 years ago?
A. No.
Q. Thank you.
A. I do not. I corresponded with those authors and we went from there, but these are the monkeys. I was talking about the rats.
Q. You were talking about the rats?
A. The rats had definite injury and the monkeys had so much scarring that a pathologist who looked at them was wondering how you could actually draw any conclusions from them. But these people did that and that's fine. That's what they did.
Q. You agree that the rats that are used in the animal studies are rats that have a genetic susceptibility to develop Mesothelioma?

A. I'm sorry. I guess -- which studies are you asking me about?
Q. Typically, the rat studies that are done involve rats. You're trying to get the disease to occur, so you use a strain of rats with a genetic susceptibility to get Mesothelioma?
A. Sometimes you do and many times you don't. I mean, the studies that Dr. Wagner did did not use particularly susceptible animals. They were just garden variety rats just like garden variety people.
Q. And the -- you would agree that, biologically, primates are far closer to human beings in their response to potential toxins than rats?
A. They're far closer. It depends on which toxin you're looking at. But, sure, as a general principle, they are closer to us. Of course.
Q. And to finish out the animal -- the other animals. Hamster studies have been done. Amosite causes Mesothelioma in hamsters but Chrysotile doesn't?
A. In that study that's right.
Q. Sir, the other aspect of your testimony related to what happens in test tubes when various substances are placed in proximity to cells; correct?
A. Yes.
Q. Actually, they're injected into the cells?

A. No. The fibers are introduced into the cell culture, and the cells actively pick up the fibers just as they do in the body.
Q. Okay. And you would agree that after this, the cell would die.
A. Very likely to die. But these studies actually go on, as we have done and others, to show that cancers develop in the dish as you're looking at it here. So that -- so they don't all die, in fact.
Q. This -- you couldn't develop Mesothelioma in the dish; right?
A. Wrong. Okay? Wrong. Because Mesothelioma is a cancer of the mesothelial cells produced by a carcinogen, and that's been done in the dish in my laboratory and a number of others.
Q. The fact of the matter is that you don't yet know what the precise genetic errors are that have to be caused in order to create Mesothelioma.
A. As I answered in examination directly to Mr.- -- to Mr. George, you're exactly right. We know a lot about how it works but we don't know the precise genes that are required. That is true.
Q. You agree that longer fibers are generally considered more potent than short fibers without question; correct?

A. Correct.
Q. You agree that the mutagenic effect of asbestos fibers at low-dose is still unknown; correct?
A. Where were we talking about? I'm sorry. A mutagen in a dish or a mutagen after inhalation? I'm sorry.
Q. What I'm talking about is what's been discussed in a conference, I think, you were involved in, the role of mutagenicity and asbestos fibers may occur in aspects of carcinogenicity and other diseases. Remember that conference?
A. Yes.
Q. All right. And you're familiar with some researchers named -- the last name, I think, is Wang. Is that correct? Well, the pronunciation may not be right. There was a publication that came out, a whole issue in the Journal of Toxicity and Environmental Health; correct?
A. Yes.
Q. All about this issue of mutagenicity; right?
A. Is that the one in 2011?
Q Yeah. Well, yes, 2011. Pretty recent; right?
A. Sure.
Q. And in Wang's article there was a discussion of "areas that require additional research," and number

three on the list was the mutagenic effect of asbestos fibers at low-dose is still unknown.

A. Right. I get the context now. That's correct.

Q. And it is correct. It is your opinion that the mutagenic effect of asbestos at low-dose is still unknown; correct?

A. I agree. Yes, sir.

Q. You spoke briefly during your exam about what may or may not happen with asbestos in the pleura, the asbestos that passes through the lung and actually makes it to the pleura. Correct?

A. Right.

Q. And, well, one other question on the mutagenic issue. The type of genetic change that occurs is based in part on the nature of the chemical reaction that occurs on the molecular level; correct?

A. Yeah.

Q. And it is a scientific truth, is it not, that the chemical nature of the Amphiboles is distinguishable substantially from the chemical nature of Chrysotile?

A. It is. That's true.

Q. Now we've heard earlier that the pleura is a structure, as displayed here on the screen, where there is fluid that runs through the area between the two layers of skin. Is that a correct anatomical

description?
A. That's fine. I described that. Yes.
Q. Sure. And what you told us is that some researchers have found Chrysotile in the pleura areas; right?
A. Right.
Q. Actually, they didn't find it in the tissue so much as they did in tumors that existed there; right?
A. Well, it was tumor tissue and surrounding tissue, I believe. And, also, I think some had found it in pleura fluid as well.
Q. And as a scientific principle, you agree that before these kinds of fiber burden studies in the pleura can tell us important information we would need controlled studies to show what the fiber levels were in healthy people or unexposed people. Correct?
A. Yes.
Q. And so far as you know, there are not a series of controlled studies that have been published on the levels of asbestos fibers in the pleura; correct?
A. Yeah. I don't know about a series, but I think normal tissues have been studied. But I don't know if there's a series that it's been done.
Q. Okay. But the kinds of fibers -- I mean we're talking about the Suzuki studies. Primarily, those are

fibers 90 percent of which are one micron in length; correct?

A. Right.

Q. They're short fibers.

A. Right.

Q. Short Fibers are ubiquitous in municipal water systems; correct?

A. They are. But they wouldn't get into the pleura tissues from the water systems.

Q. Well, they get into the body through the water?

A. Sure. They're ingested.

Q. And autopsies are done with regular street water; correct?

A. They're ingested. You're talking about from the water system. They're ingested. No one -- I can't imagine how you get -- how you get fibers in the -- in mesothelial tissue or in the pleura by ingesting fibers.

Q. Sir, it is correct, is it not, that the Suzuki studies were funded by a Hawaii plaintiff's lawyer without attribution? Correct?

A. I don't know that.

Q. You don't know about -- well, okay. And that those studies don't have a series of -- most of the time when they're published -- well, let's just go on past those.

It is true, is it not, that there are published studies that show that Amphiboles reach the pleura in significant quantities; correct?

A. Sure, they do.

Q. Sure. And Dr. Welch -- Dr. Weill explained to us that the structure of the pleura is such that short fibers can pass through the pleura and exit through the lymphatic system. Do you agree or disagree with that proposition as a scientific fact?

A. No, I agree.

Q. And do you agree or disagree that the long fibers have much more propensity not to be able to get out through the stoma, out of the lymphatic system?

A. True.

Q. All right. And that has been published by Donaldson in 2010; correct?

A. That's one of the places. Yes.

Q. You agree, sir, that the concept of a threshold may depend on fiber type, correct, and whether it's Chrysotile or crocidolite or amosite?

A. No. I don't understand that. Because a threshold is the level below which you -- above which you're trying to find an effect. So that could be for any fiber type.

Q. Well, maybe it isn't.

A. Maybe I didn't understand your question.

Q. Maybe I didn't ask it correctly. Let me repeat the question. You agree that the concept of a threshold may depend on the fiber type, whether it's Chrysotile or crocidolite or Amosite?
A. I'm sorry. The concept of a threshold is the ability to be able to find a level above or below which you can find an effect. But that concept would be the same whatever the fiber type.
Q. Well, sir, I don't mean to argue with you.
A. I don't want to argue either.
Q. We know that something different's going on with Chrysotile; right?
A. I don't know what you mean by "something different." I mean, please --
Q. Sure. I'll be more clear. You were asked in your deposition in this case, "Do you agree that the concept of a threshold may depend on the fiber type, whether it's Chrysotile or crocidolite or amosite?" Correct? And you said, "That may be true."
A. Well if the concept is the level, is the threshold, then, sure. But if the concept is establishing a threshold, then I don't see any difference in how you do that.
Q. Back to the issue of the Suzuki studies. Do you agree that autopsies are done with regular municipal

water usually?
A. I would think so. Yes.
Q. Yeah. And the samples that Suzuki was looking at were samples that were harvested in typical autopsy processes; correct?
A. I think so.
Q. Okay. What you've told us, sir, is that basically you -- that the precise mechanism is not understood and there are a number of different theories about how asbestos fiber types may induce Mesothelioma; correct?
A. So I told you the levels at which we do know and don't know the answers to certain questions. And I guess you'd have to go on then with the next part of your question.
Q. Okay. Do you agree, sir, with Dr. Mossman that it's a very complex issue, this mutagenesis issue, and that we don't yet even know if we're dealing with one kind of tumor or several types of tumors?
A. That's fine.
Q. By "that's fine," you mean you agree?
A. Yeah, I agree. That's fine.
Q. Sure. Sir, let's see. We've heard a lot in this case about OSHA regulations and various protective procedures instituted at various times, including a few years ago. You agree, sir, that the people that write

OSHA regulations and public health agencies have a charge to be protective of public health; right?

A. Yes.

Q. And they want to build in a safety margin in any of their safety standards.

A. Well I would think so, but I'm certainly not conversant with the things that OSHA does and says.

Q. We've heard that public health agencies look at data that has accumulated in what's called a zone of observation and then they make projections to a zone of inference. Are you familiar with that fact?

A. No.

Q. Then we'll go on.

So, we have theories. You -- the lawyer here has called it plausibility. It's plausible, for example, that cigarette smoking causes Mesothelioma; right?

A. That would be plausible. And that was tested and found not to be true, but it's plausible. Sure.

Q. And it's plausible because lots of cases have been reported of Mesothelioma among people who have an occupational history of exposure to cigarette smoke; right?

A. Right.

Q. We could get a case series of 30,000 cases like that if we wanted to go through the literature; right?

A. Right.
Q. All right. And, of course, it's clear that there are lots of carcinogens in cigarette smoke; right?
A. Sure.
Q. And clear that they get to the pleura.
A. I'm sure. Well, actually, I don't know that that's clear actually. In fact, that might be the reason that cigarette smoking has nothing to do with Mesothelioma, that the carcinogens may not get to the pleura. I don't know if that's been established.
Q. Have you seen the -- well, let me just show you -- I think we have a picture of -- I don't have it here. We showed a picture earlier of the black spots that exist on the pleura, and the explanation was that tars from cigarettes accumulate in the stoma.
A. Well, yes. I've seen that, actually. You actually gave an outline, a black outline around the --
Q. The tars in cigarette?
A. I'm sorry.
Q. Go ahead.
A. We don't know that those are carcinogenic. In other words, we don't know that -- if, in fact, those were carcinogens that were collecting in those areas I can't say, but I'd expect there to be high rates of Mesotheliomas.

Q. But just because a carcinogen reaches the pleura tissue does not necessarily mean that that carcinogen produces the rare type of cancer we call Mesothelioma; right?
A. That's why you have to do all the studies that allow you to draw the conclusions. And the studies asking if cigarette smoke causes it say it doesn't.
Q. And the studies as we've heard were epidemiological studies; right?
A. Right.
Q. And the epidemiology demonstrated that despite the theories, some of the associations were a little above one, some below, but none were statistically significant. And generally, they were around or lower than one so that we knew cigarette smoke is not a cause of Mesothelioma; right?
A. Yes.
Q. You would agree that the acid test of who gets the disease and what causes it is epidemiology, of course. Right?
A. Yes.
Q. These are your words: So whatever theories we might have we've got to look at the epidemiology.
A. Right.
Q. Now you started the exam -- the exam was started

-- you have no control over the questions that are asked, I understand -- with a discussion of the Bradford-Hill criteria.

Q. And you are aware sir -- well, let me just go back to the document that Mr. George -- Mr. George read to you.

MR. GEORGE: Your Honor, I have no objection to Dr. Brody answering questions about the Bradford-Hill criteria. Again, I would object to him interpreting what the legal significance of that is.

THE COURT: All right. We'll let him testify.

BY MR. SCHACHTER:

Q. Sir, Mr. George read to you this paragraph from the address that Sir Austin Bradford-Hill gave, I think it was in 1965. Is that what we -- you read at the beginning of the examination?

A. Yeah.

Q. Okay. And that comes in the article, as you know, after Sir Austin Bradford-Hill has listed his nine criteria. Correct?

A. Right.

Q. And in what you read, it's clear that he asks did -- he says, there are nine viewpoints from all of which we should study association before we cry causation. Correct?

A. Yes.
Q. Before listing his criteria, is it true that he wrote, disregarding, then, any such problem in semantics we have this situation. Our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation? Did that -- is that how he introduced his criteria?
A. Right.
Q. Okay. Would it be a fair construction of this document that from a scientific point, as you view the scientific literature, Sir Austin Bradford-Hill was saying, okay. I've got an association perfectly clear-cut and beyond what we attribute to the theory of chance. Now I'm going to apply these criteria?
A. That's what he's saying.
Q. And you agree, sir, that the way epidemiology works, to the extent you understand it, is that studies are done to determine whether the relative risk is statistically significant. Correct?
A. Well, that's the way epidemiologists do it. But, I mean, typically -- but I'm not an epidemiologist. So you'd need to ask an epidemiologist if they do that all

the time. But, I mean, also, Dr. Hill, Bradford-Hill, also said in that article, if I remember correctly, that he wouldn't require a statistical test. I believe there's a paragraph in there that says that. So he's relying more on the individual criteria that he uses, rather than a statistical test.

Q. Sir, the best paragraph for your side was read at the beginning of your examination and it says what it says.

MR. GEORGE: I would object to the characterization of the testimony and move to strike.

BY MR. SCHACHTER:

Q. I apologize. Sir, if we look at how epidemiology works. Your example, smoking and Mesothelioma, are the examples, smoking and Mesothelioma shows it's a search for a statistically significant increased relative risk. Correct?

A. I agree.

Q. Okay. Now just so there's no doubt, you would agree that Chrysotile differs chemically from the Amphiboles. Correct?

A. Yes.

Q. It differs electrically from the Amphiboles?

A. Yes.

Q. It has a shorter duration in the body.

A. Correct.
Q. It's structurally different. It's curly, as opposed to spear-like or straight, which the amphiboles are.
A. Many of the fibers are. Yes.
Q. And they're easily broken.
A. Correct.
Q. We looked at the chemical formulas and they're completely different. Not completely, but substantial differences in the chemical formulas. Right?
A. True.
Q. You've talked to us about your background. You studied with Dr. Wagner first; is that correct?
A. Yes.
Q. And that was the famous Dr. Wagner who in 1960 had the case series that talked about the probable connection between crocidolite and Mesothelioma; correct?
A. Right.
Q. And later, a number of studies were done and confirms the association between crocidolite and Mesothelioma; right?
A. True.
Q. All right. And ultimately, he -- and he did these animal studies, these rat studies, some of them that were the first rat studies that were published. Right?

A. Correct.
Q. He ultimately concluded that Chrysotile was not a cause of Mesothelioma in human beings; correct?
He ultimately concluded crocidolite was the only asbestos that causes Mesothelioma. So he refuses the amosite data and Tremolite data. So in other words, at some point in his career he -- oh, I'm sorry Your Honor (cell phone rings). It's off. I apologize.
THE COURT: That's okay.
THE WITNESS: Where was I? Okay.
BY MR. SCHACHTER:
Q. You were explaining to us that Dr. Wagner not only didn't believe the that Chrysotile was the cause, but he also had some questions about amosite. Right?
A. Yeah. Right. Well, he said he had this crocidolite hypothesis that it was the only cause. I don't know any other scientist that thought that way. I don't know why he changed his mind, but he precipitously did so. So, you know, one could guess, but I'm not sure why.
Q. Okay. We don't want your guess, sir.
A. Yeah, right. So Dr. Wagner, when I worked with him, showed me that Chrysotile causes Mesothelioma.
Q. And you formed that opinion in the '70s; right?
A. Right.

Q. And after that, I guess starting in the '80s, you began testifying for, primarily, plaintiffs in the asbestos litigation. Correct?
A. In the -- yes '89, early '90s. Yes.
Q. And as new literature developed, you didn't change your opinion on that; right?
A. No. Because the new literature was quite clear in supporting that opinion that Chrysotile causes Mesothelioma.
Q. Your view of the literature is that it supports that, sir. Right?
A. My view of the literature is that it supports it. That's fine.
Q. All right. And you studied also under another very famous researcher named John E. Craighead; correct?
A. Right.
Q. And he is the author of a 2008, or the editor of the Oxford University Press book Asbestos and Its Diseases. Correct?
A. Yes.
Q. And you still consider him a very fine scientist?
A. Sure.
Q. And he's your mentor or one of your mentors?
A. He was. Yes.
Q. And he has reviewed the evidence as it has evolved

and is of a view that the evidence is totally convincing that Chrysotile does not cause Mesothelioma. Right?

A. That's his view.

Q. All right. And now we're going to a talk and focus in on this case. We've talked about the thousand fiber per cc miners, and I'm sure we'll hear more about mining populations. But you would agree that it is the consensus of the medical community that Chrysotile-induced Mesothelioma only occurs with very high exposure. Correct?

A. Well, you read my answer. I would, as a general principle, think that it's true. I mean that's where most of the cases come from, but there are numerous reports of cases from low-dose exposures

Q. We have case reports and we'll deal with case reports. But you agreed in your deposition and still agree, sir, that if we're looking for a consensus in the medical community, it's that Chrysotile-induced Mesothelioma only occurs with very high exposures. Correct?

A. No. Where does it say "only?" "Only occurs?" That's where most -- that's where most of the cases come from are from high exposures. That's what that says.

Q. Let me just make sure your answer is in the record. The question was, at your deposition would you

agree that it is the consensus of the medical community that Chrysotile-induced Mesothelioma only occurs with very high exposures? And your answer was, I would as a general principle -- I think that's true. I mean that is where most of the Mesothelioma is caused by Chrysotile come. Right?

A. Exactly. That's not where they all come from. That's where most of them come from.

Q. In fact, that is published in a book that is authored by a very famous scientist, including physicians at the Mayo Clinic. Right?

A. Yes.

Q. And when they do it, they go on to say that it's only at very high exposures. And they talk about where that's been shown as being in the mining situations where there is a very high level of asbestos that can be found in the miners' bodies. Right?

A. That's where most of the cases come from. That's right.

Q. Sir, we talked a little bit about potency. There have been researchers and published health analysts who have looked at the level that even assuming that we're dealing with Chrysotile in the mining situation what the potency is on a fiber per fiber basis. Correct?

A. Yes.

Q. Now in this case, at great expense, we've had a lot of information developed about people who may make claims against Garlock in the future and they've been grouped into various categories. I'll represent to you that those group groups have been identified to the Court and a retrospective exposure assessment has been done using the methodologies for doing that kind of work and that even in the highest exposure category the relative contribution in terms of fibers from lifetime cumulative exposure is shown. For the pipefitters here it would be 5.5 in one year, 5.5 fibers per cc year as opposed to gaskets, even if you assume three a day. I think it's three a day.

A. You know, I'm sorry to interrupt. I have no idea what you're talking about.

MR. GEORGE: I was going to object to the foundation.

THE WITNESS: Why are you showing me this?

MR. GEORGE: He's a cell biologist.

BY MR. SCHACHTER:

Q. Yeah. You have testified, sir, that asbestos -- that amosite is 500 times more potent on a fiber-per-fiber basis. Correct?

A. Okay. That's not my testimony that it is. It's my testimony that there is a scientist Hodgson and

Darnton who said crocidolite was 500 times and amosite is 100 times. Yeah, sure, that's fine. I don't think there really is a good number because they changed their numbers after that.

Q. Okay.

A. So, please, go ahead I guess.

Q. Thank you. You testified before a jury in a trial in Boston, and I believe this happened in 2006. Right?

A. Whatever the date is.

Q. 2006. Now we've heard some testimony in this case, or seen some articles written before 2006 by Nicholson and Boffetta and Allan Smith. And you were aware of all that literature. You're up on the asbestos litigation; right?

A. Mostly, yes.

Q. All right. So that was all in the literature as of 2006. And you testified, did you not, to this 500 number. And you mentioned Hodgson and Darnton and others. But the lawyer who was asking you questions, and it wasn't me. You said in answer to one of them that 500 was my number. And the lawyer asked you, that is your opinion? And you said, that's my opinion. Correct? The difference between amosite and Chrysotile was 500 that you testified before that jury in 2006. Right?

A. Okay. That's fine. I think it was 500 for

crocidolite, actually, on that paper, and 100 for amosite. But I'm also testifying today that they changed those numbers. And I don't think those numbers are really mean very meaningful, if the authors who put them together have changed them.

Q. Sir, even in this case when I took your deposition you said that -- you said there were a lot of numbers in the literature, but you said that the 500 number is a good number.

A. I'm sorry. I said?

Q. Huh?

A. I'm sorry. What did I say?

Q. Just a second. Let me get your testimony. This is from your testimony a few months ago. Isn't it true that you previously testified that amosite is 500 times more potent than Chrysotile in causing Mesothelioma? And your answer was, on a fiber-per-fiber basis, absolutely. And let me get the next pages. It went on for a while. And you made these explanations about the Hodgson and Darnton --

A. Can we see that? Actually, I think I just said the same thing.

Q. Here?

A. No. I mean put it under the thing there so we can see it. It's exactly what I just said. "Okay. All I'm

trying to point out is that my testimony yesterday is under oath and today under oath." Next page.

Q. (Indicating.)

A. "That the Amphibole fibers are more potent on a fiber-per-fiber bases. That's all it means, whether it's 500 times, which I've testified to and I agree with, or two times in some studies, 800 times in others. On a fiber-per-fiber basis what that means is you may need 500 Chrysotiles for every Amphibole. That is fine. It depends on what the person is exposed to. That's all I want the jury to understand."

So, I'm -- the 500 times number by these authors Hodgson and Darnton has been changed. So you show -- you can show me this, which is exactly what I've told you, but these numbers have changed. Please go ahead.

Q. Okay. And buried in all that explanation, you ultimately said 500 is a good number. Right?

A. Well within the explanation that I gave for the numbers? Sure.

Q. What you're referring to is --

A. It's no better than -- I'm sorry. It's no better than two times. It's no better than 800 times because we don't know the number.

Q. You are -- sir, you agree that there's reputable scientific evidence that Chrysotile is far less potent on

a fiber-per-fiber basis than amosite in causing Mesothelioma?

A. Yes.

Q. And that's a consensus of the scientific community is that the causes of Mesothelioma at all, Chrysotile is far less potent?

A. Yes. And I don't think we know just how much the differences in potency are.

Q. But, for example, if we had a pipefitter -- if we look at the pipefitter numbers before this court. As a scientific principle, it would be important to include potency, a potency factor, in any of the exposure that would be attributable to the Amphibole containing insulation; correct?

A. Sure, if you knew what it was. If you didn't know what it was, I don't know how helpful that would be.

Q. All right. And if in fact those numbers that we gave to the Court were to take into account your potency limit at only 50 times one order of magnitude less, it would greatly swell the relative contribution of the component of the exposure that was from insulation. Correct?

A. Just as a general principle? Sure. But I can't speak to what's in the insulation to begin with.

Q. Thank you, doctor.

A. You're welcome.

REDIRECT EXAMINATION

BY MR. GEORGE:

Q. Some quick followup. Dr. Brody, I'm just going to go from the front back. I want to start -- what I want to start with is, I want to ask you about -- Mr. Schachter put up a slide that talked about the studies of the animals. One of the studies he put up was the Stettler study, the slide he said was monkeys, amosite get Mesothelioma and Chrysotile don't. But the Stettler study has nothing to do with Mesothelioma; correct?

A. Well this says Histopathology and that's why I responded by saying I looked at those monkeys and they were a mess.

Q. What this was, they did a chronic exposure for 18 months. They exposed rats and monkeys to a level of about 0.79 fibers per cc. They sacrificed the first group by 24 months. That's the paper that Platek wrote?

A. Right.

Q. Then they went back 11 and a half years later and looked at the surviving monkeys and sacrificed them to see what was happening.

A. Right.

Q. Now, one thing we know is that there was only an

11-year latency period.
A. Right.
Q. Would we expect that if they waited longer there would be more effect from exposure?
A. Could very well be sure.
Q. Do we know what the latency period is in a monkey?
A. We don't.
Q. They say that -- the other thing that he didn't mention is when they did this study they used short Chrysotile which had been prepared by ball milling. Do you know what ball milling is?
A. Yes.
Q. Can you explain what ball milling is?
A. Well, when you take a sample of asbestos you can break it up using a large ball and it breaks it down into smaller fibers, mills it.
Q. Does that have an effect when an animal inhales ball-milled Chrysotile as opposed to inhaling regular Chrysotile?
A. Well, there are shorter fibers. But, obviously, in the studies I've done, and this study, they cause disease. Short fibers cause disease.
Q. And what the authors found is the actual authority, short fiber exposure in the present study was quite small. But they go on to say 52 percent of all

particles examined by scanning electron microscope -- and that's what you use; right?

A. Right.

Q. Were non-fibrous, primarily clumps of small Chrysotile fibers produced by ball milling of bulk Chrysotile. These clumps of Chrysotile remained in tact in the SEM microcongraphs of the rat lungs. In addition, it should be remembered that short Chrysotile was prepared by ball milling. Other investigators have noted that mechanical milling changes the crystalline structure and the surface chemistry of Chrysotile, since service cell chemistry is thought to play an important role in fiber-related lung fibrosis and carcinogenicity may have affected the fiber. It should be noted that the design of the present study allowed for only a small number of animals and low exposure levels and duration relative to human exposures. Hence, the ability to draw inferences from this data is limited.

Do you agree with that?

A. Sure.

Q. He also talked to you about the baboon studies. Now, he didn't tell you that in the Goldstein baboon study they had no idea how much exposure that the Chrysotile baboon had in relation to the exposure that the other baboons had. If that baboon had less exposure,

would that be a reason why maybe that baboon did not develop Mesothelioma?

A. Sure.

Q. If the baboon had equal exposure and if Chrysotile is more potent, then we wouldn't expect Chrysotile to be induced on equal exposures of Chrysotile for crocidolite. True?

A. Sure.

Q. If we wanted to find out if Chrysotile causes Mesothelioma in baboons we would need to give them more Chrysotile than amosite and crocidolite. Correct?

A. Sure.

Q. And then in the last study, Hiroshima, they gave the baboons Chrysotile for eight and a half to 24 months. But they gave the amosite for 49 months and the crocidolite for 35 months, which was the dose that produced the Mesothelioma. Would the fact that there was less exposure from Chrysotile be an explanation of why the baboons did not get disease?

A. Yes.

Q. Are you familiar -- you were asked some questions about iron. Are you familiar with the paper that just came out in 2012 on iron overloads signature in Chrysotile-induced malignant Mesothelioma?

A. Yes.

Q. And they found -- in fact, they did exposures. They produced Mesotheliomas with all three types of asbestos and they said that these data indicate that Chrysotile is a strong carcinogen when exposed to the mesothelia acting through the induction of local iron overload. What does that mean?

A. Well all the asbestos varieties generate oxygen radicals, and the iron in the fibers is the catalyst for that. And some people think that Chrysotile might have lower potency because it has less iron but, in fact, we've known that it accumulates iron over time. And that's really what that paper addresses. It accumulates iron and can, therefore, generate oxygen radicals and act as a carcinogen. We published in my laboratory in 2004, the fact that Chrysotile asbestos generates oxygen radicals and, therefore, is part of the carcinogenic process.

Q. If it's a carcinogen when exposed to mesothelia, does that mean it's capable of producing a Mesothelioma, which is a cancer of the mesothelial cells?

A. Yes.

Q. You were asked about the Hill causation. And you had said you thought you remembered a section about test of significance. Is this the section that you remember?

A. Yes.

Q. It says that no formal tests of significance can answer those questions. Such tests can and should remind us of the effects that the play of chance can create, and they will instruct us of the likely magnitude of those effects. But beyond that, they contribute nothing to the proof of our hypothesis. Does that mean that Hill's requiring that you have a doubling of the risk with a statistical significance, not including one?

A. No.

Q. You talked about the fact that Hodgson and Darnton's numbers of 500 to 100 to one. You were asked about that at trial in 2006?

A. Yes.

Q. That was seven years ago; right?

A. Yes.

Q. In 2010, four years after that testimony, Hodgson and Darnton wrote a letter to the editor saying based on the new data coming out of North Carolina we were off by a factor of ten. Is that right?

A. Yes.

Q. Would the fact these authors who came up with the ratio of 500 to 100 to one changed it by a magnitude of ten alter your opinion as to what their belief is as to potency?

A. Right. Which is why Mr. Schachter and I went

through that about what is really a good number and what isn't.

Q. If we reduced by a magnitude of ten, we're really talking about a ratio of 50 to ten to one, right?

A. Yes.

Q. Thank you very much.

RECROSS EXAMINATION

BY MR. SCHACHTER:

Q. Jiang was a rat study?

A. I'm sorry?

Q. Jiang was a study in rats, the study he showed you there. Do you know or not know?

A. I don't. I don't remember. This one we just saw, you mean?

Q. Yeah, the Jiang.

A. Yes.

Q. And the Hodgson and Darnton point is -- what they did was they said that the data in Loomis varied by, from their numbers, by a factor of ten. Correct?

A. I don't remember them saying that. But if that's what it says, fine.

Q. And Loomis is the article co-written by Dr. Dement and containing data on the Marshville plant; is that correct?

A. That's what I understand.

Q. All right. We'll hear more about that later. Thank you, sir.

A. You're welcome.

THE COURT: Okay. You can step down. Thank you.

(Witness excused at 3:18 p.m.)

THE COURT: Why don't we take a break now and come back at 3:30?

Let me -- just housekeeping-wise, let me say Mr. Moon you may want to listen to this because I don't see Mr. Swett or Mr. Inselbuch. Somebody has filed a motion with respect to the confidentiality. So if you are you all aware of that?

MR. MOON: I've seen it.

THE COURT: I won't do anything about that until tomorrow -- maybe tomorrow morning. If you all want to file something in response to that, if anybody wants to file any response to that, do so as soon as you can. We'll deal with that in the morning, probably without a hearing. Okay? Thank you. Be back at 3:30.

(Off the record at 3:19 p.m.)

(On the record at 3:32 p.m.)

THE COURT: Have a seat. Let's go on to whatever is next.

MR. FROST: Thank you, Your Honor. We'd call Carl Brodkin.

(Witness duly sworn at 3:32 p.m.)

DIRECT EXAMINATION

BY MR. FROST:

Q. Good afternoon, Dr. Brodkin. Can you please state and spell your name for the record?

A. Carl Andrew Brodkin. C-A-R-L. A-N-D-R-E-W. B-R-O-D-K-I-N.

Q. And Dr. Brodkin, what is your specialty?

A. I'm a physician in Occupational and Environmental Medicine and internal medicine.

Q. And just briefly, what areas of expertise does it take to be a specialist in Occupational and Environmental Medicine?

A. As in other branches of medicine, one has to complete medical school, a residency training program with internship and residency, and then advanced training for fellowship.

THE COURT: The first thing you've got to do is pass chemistry, which is what I couldn't do.

THE WITNESS: Yes, Your Honor. Yes. Organic chemistry is the weed-out class.

BY MR. FROST:

Q. I couldn't pass that either. Maybe that's why we're lawyers.

Dr. Brodkin, where did you graduate? And when you

graduated, did you receive any honors?
A. I attended Swarthmore College where I got my bachelor's degree. I then went to my home state of Colorado to the University of Colorado for medical school. I graduated with honors from medical school.
Q. And in fact, Dr. Brodkin, weren't you in the top of your class in medical school?
A. Well, I was elected to Alpha Omega Alpha which is the Honoured Medical Society.
Q. Now I have a slide here that just has some of the things so we can talk about these briefly. I think you just talked about the Alpha Omega Alpha. Have you ever done any fellowships and worked with the ATSDR?
A. Yes. After I completed fellowship training in Occupational Medicine I did another year of fellowship training in Environmental Medicine with ATSDR.
Q. And I have the American college of Occupational and Environmental Medicine. How does that play into your background and experience?
A. The American College of Occupational Medicine is the largest body in the U.S. of Occupational Medicine physicians. I've been a member for almost 20 years since I've completed my fellowship in Occupational Medicine. I'm a fellow of the college and also served on the Lung Disorders Committee of the American College.

Q. And are you board certified in Occupational Medicine?
A. Yes. After I completed internal medicine I became board certified in that specialty. And after I completed specialty training in Occupational Medicine I became board certified in that area.
Q. And I have the Fred Hutchison Cancer Research Center. How does that play in your background?
A. In my fellowship in Occupational Medicine I became interested in asbestos-related disease and became a co-investigator of a large cohort of asbestos-exposed workers, over 4,000 workers, and that was organized through the Fred Hutchison Cancer Research Center. It was a large National Cancer Institute study to look at risk factors and the development of cancer and antioxidants and vitamins that may prevent those cancers. I had the opportunity to follow those workers for more than 17 years. They're still being followed.
Q. And in fact, have you given safety lectures in the past?
A. I have. Certainly, local unions have asked me to participate in lectures. I gave the Dunn Memorial lecture for the Pipefitters Local in northern Oregon and southern Washington, my home state now, about health and safety issues.

Q. And so you've had experience with not only talking to pipefitters but, also, in some of your studies you have followed people who were pipefitters and those type of people?

A. Yes. Where I practice in Seattle, there's a large shipyard industry. And I've seen thousand -- of the thousands of workers I've seen over the years, probably about a third of them at some time have worked in the shipyard industry. So I'm familiar with that and with the CARET study through Fred Hutchison. Of those 4,000 workers, almost 1,000 of them, or a quarter, were pipefitters.

Q. And in fact, I have a slide up there that has the CARET study. Just briefly, what was the CARET study?

A. The CARET study was a prospective randomized epidemiologic study to look at whether antioxidants and vitamins prevented cancers in high risk individuals, including smokers and asbestos-exposed workers. The asbestos-exposed cohort was over 4,000 workers and allowed an opportunity not only to look at whether the vitamins were effective but, really, at the natural history of asbestos-related disease and the development of malignancies in that group.

We stopped the trial in 1996 because the vitamins, unfortunately, were not effective in reducing cancer

rates. But we were able, certainly, to follow those workers and do many publications and have increased knowledge regarding asbestos exposure which have come from the CARET study, and I participated in a number of them.

Q. And I have up there pipefitters. Were they part of this group? And how many folks were part of it?

A. They were. Pipefitters were the largest group, about 1,000, a quarter of the workers. The others included other shipyard trades. There were over 700 boilermakers, about 250 insulators and some other shipyard trades, as well as plasterboard workers.

Q. So there were more pipefitters in your study than even insulators?

A. Oh, yes.

Q. And so that was a large group in this study group that you had worked with?

A. Yes. The insulator unions are -- tend to be much smaller in our area than the pipefitters in terms of numbers, so that's reflected in the CARET study.

Q. And where you have up there, "pipefitters 40 cases of Mesothelioma." Among those thousand workers there was at least 40 cases of Mesothelioma?

A. Well, 40 cases occurred in the cohorts. So, among the 4,000.

Q. I'm sorry?
A. Among the 4,000 workers there were 40 Mesotheliomas. That's about one percent of the population. So of the cohort, for a rare disease like Mesothelioma which occurs in about one in a million individuals, it's an extremely high rate. There were about 11 Mesotheliomas in the pipefitters. So, over one percent.
Q. So 11 of the 40 were in the pipefitters?
A. Eleven of the 998 were among the pipefitters.
Q. Okay. Now -- and you mentioned this briefly. But that CARET study and this characterization of asbestos, this -- as an asbestos cohort, you've actually published this with other authors?
A. Yes. There were a number of co-investigators. I was one of this group of co-investigators, and this is one of the publications that characterizes the types of workers and how workers entered the CARET study.
Q. And there were other articles that were -- this same group of people, but there's multiple articles that you were a co-author on dealing with this study. Correct?
A. That's correct. Most of my 50 -- approximately 40 to 50 peer review publications are related to asbestos, and many of them are through the CARET study.

Q. Now -- and we'll get to a few of these. Was this one also related to the CARET study, the one with Harvey Checkoway?
A. Yes. This was looking at the correlation between respiratory symptoms and lung function between asbestos-exposed workers.
Q. Now you mentioned just briefly you have published on asbestos. And all the articles that I just showed the Court, those are all peer reviewed and published in the literature. Correct?
A. That's correct. Yes.
Q. Besides that, you've also published other things concerning asbestos. Is this one also -- this one is also related to the CARET study; correct?
A. It is. This is looking at lung function changes over time in asbestos-exposed workers and what would predict loss of lung function.
Q. Now, besides the CARET study, have you also, in your personal practice, seen people that suffer from asbestos-related diseases working in the Washington state area?
A. Yes. As I have said, since my fellowship beginning in 1989 I've seen asbestos-exposed workers. And in clinic or in surveillance programs or in the CARET study, I've seen thousands of asbestos-exposed workers

over the last 20 years. And that's certainly gone on throughout my practice, both when I had an academic practice at the University of Washington as well as in private practice since 2003, although my practice now is mostly a consulting practice.

Q. Now you've also -- I have a textbook. And I didn't bring it, because it's pretty big. It's about this thick. Are you an editor of the Textbook of Clinical, Occupational and Environmental Medicine?

A. Yes. I was one of the co-editors of the second edition of that textbook.

Q. Now, besides that, you mentioned briefly that you've worked at the University of Washington. What have you done teaching in the past?

A. Yes. I was a full-time faculty member for about ten years after completing my fellowship, and I continue to serve as an adjunct clinical associate professor. But in those ten years I was at the university, I was variously at different times residency director, clinic director and course director for their clinical Occupational Medicine course.

Q. And have you also -- other than your peer reviewed articles and your editoring of the textbook, have you been asked at times to be on committees such as the American Thoracic Society?

A. Yes. I certainly have been on a number of committees. In the early 2000s I was asked by the American Thoracic Society to participate in a committee that would advise pulmonary physicians, as well as physicians in internal medicine and other specialties, about the criteria necessary to diagnose asbestosis, the scarring disease related to asbestos, as well as pleura plaques. I'm not a lung specialist but was asked because I was an Occupational Medicine physician experienced with asbestos to participate on that committee.

Q. So even though the American Thoracic Society is a society for lung specialists, your specialty is Occupational Medicine, and you were still asked to be on this committee that came up with the criteria of how to diagnose asbestosis in individuals?

A. That's correct. And this became the consensus document for the American Thoracic Society on how to diagnose asbestosis.

Q. And there was a number of folks that were on that committee?

A. That's correct.

Q. Now, the other thing about this is that these consensus documents -- the Court's heard a little bit about consensus documents. Based on your experience being involved in the ATS consensus document, how does

that process work?
A. The process works, typically, by selecting a committee of experienced physicians and scientists in an area to develop the criteria to provide evidence of scientific and medical reliability and validity to produce a document that synthesizes that review and then to present it to the broader organization for review to see if there is consensus. If there's not, one may have to go through an iterative process. But in this case, certainly, our document went through the broader committees of the American Thoracic Society before it was adopted as a consensus document.
Q. Now I didn't ask you, but have you had any involvement with the American College of Occupational and Environmental Medicine?
A. Well, I did speak to that a little earlier that I've been a fellow with the American College of Occupational and Environmental Medicine and do serve on the Lung Disorders Committee and have for a number of years.
Q. What's the Lung Disorders Committee?
A. The Lung Disorders Committee is the committee of the American College that really advises their board of directors on issues relating to pulmonary disease in occupational settings and positions that might be taken

based on evidence, scientific evidence, or developments in the field.

Q. Your Honor, we would offer Dr. Brodkin as a expert in the area of Environmental Medicine.

M. SCHACHTER: No objection.

THE COURT: All right.

MR. FROST: And occupational.

THE COURT: And occupational?

M. SCHACHTER: No objection to that either.

THE COURT: He will be accepted.

BY MR. FROST:

Q. Okay. Dr. Brodkin, how much do you get paid an hour?

A. I'm getting paid $550 an hour, which is my 2012 rate when I was retained in this case.

Q. Now how many hours have you spent on this case, I guess, prior to your deposition?

A. Over 100 hours.

Q. And that hundred hours would be billed at your normal rate, I guess?

A. All of my activities in this case and other evaluations I do are billed at an hourly rate.

Q. Okay. Now, we want to talk about -- we talked with Mr. Templin a little bit and about state of the art and asbestos and gaskets and packing in particular. Is

that an area that you have reviewed the literature on?
A. It is. It's certainly a common exposure in Occupational Medicine, and I have.
Q. And as an Occupational Medicine doctor, this is something you need to review in order to have an understanding of not only the work practices but what was known and knowable about these type of products over the years?
A. Certainly one has to be aware of the literature in terms of understanding both exposure, because Occupational Medicine is an assessment of exposure-related illness. So the literature regarding exposure, as well as the literature regarding health effects. Through my review of that literature and studying it, I am aware of how medical knowledge did evolve over time.
Q. And the first document I have is Merewether and Price from 1930. Are you familiar with that particular document?
A. I am. Yes. Certainly, we were taught about this when I was a fellow.
Q. So this is something you were actually taught in school?
A. Within the first couple of weeks of fellowship in 1989 when I started Occupational Medicine, this is one of the seminal papers in all of Occupational Medicine.

Q. This Merewether and Price paper. Mr. Templin talked about the fact it was in two parts. He and I talked about one section, but I want to talk to you about other areas. Was Merewether and Price just focused on asbestos, Chrysotile asbestos, textiles in England? Or were there other issues being discussed concerning asbestos products?

A. The initial study was an investigation of textile workers who were Chrysotile-exposed. Merewether and Price identified about a quarter of those workers that had asbestosis. And based on the realization that there was prevalent disease among asbestos-exposed workers, they made recommendations for other workers in other areas. That's part two of their document where they make those recommendations.

Q. And did they talk about any end products that were not asbestos textiles?

A. They reviewed a number of different products, including insulation products, cementious products like asbestos cement, friction products, jointings and packings, which was an older name for a gasket-like material. These were all areas that they studied.

Q. And in the 1932 -- this is what I talked with Mr. Templin about briefly. In 1932 did they continue to talk about packings and jointings?

A. They did, although they had broader recommendations in 1932. 1930 was strictly focused on workers fabricating or manufacturing materials. In 1932 they focused not only on that group of workers but on industrial processes. So that really refers to workers that would be using the materials in industrial processes, including end users.

Q. And so after 1932, did the knowledge or at least understanding that individuals could get asbestosis from jointings and things like that continue on?

A. It did. And certainly it was recognized that activities disrupting the material, specifically sawing, grinding and turning of a packing and joining material and other material could result in exposure that puts workers at risk for asbestosis.

Q. And I put up there, again, another article from 1935. That was, again, from the Merewether and Price; correct?

A. Yes.

Q. Then in the same time period of 1935, were there other documents from the United States, particularly the Pennsylvania Department of Labor and Industry where they also talked about asbestos and concerns for end products?

A. In 1935, Campbell essentially replicated the Merewether and Price study in textile workers in the

United States and found a very similar prevalence, about a quarter of workers in the textile trades with evidence of asbestosis. And very much like Merewether and Price, the Pennsylvania Department of Labor and Industry document in 1935 made specific recommendations for different types of materials which certainly included a rope and wick-type materials, packing-type materials that can contain up to 90 percent asbestos. It would also include friction products, insulation, cementitious materials very much like Merewether and Price.

Q. So, again, we're talking about not only the people manufacturing products but concern for end products?

A. That's true. Yes.

Q. Then I have a document entitled Occupational Tumors and Allied Diseases. Now this isn't just a paper, Dr. Brodkin, this is actually a big old book. I mean, that's what I'd call it. It's about two or three inches thick. And it's a book published by Dr. Hueper who we talked with Mr. Templin about in some of the internal Garlock documents.

A. Yes. Occupational Tumors is a text. It's over 500 pages long. And Dr. Hueper was probably the preeminent occupational pathologist of the time.

Q. And that was published in 1942.

A. That's correct. Yes.

Q. Now, this idea, this concern, about asbestos causing cancer, in particular lung cancer. Was that something Dr. Hueper talked about in that particular book?

A. It is. Certainly, there was knowledge from the 1930s that asbestos-exposed workers were at increased risk for lung cancer. So Dr. Hueper not only addresses asbestosis, which Merewether and Price did, but also the concern for lung cancer in terms of a range of asbestos products.

Q. And what types of products was Dr. Hueper talking about as potentials for exposure?

A. It would be probably a broader range than Merewether and Price but certainly would include the same major categories of asbestos cement, insulation, friction products, gaskets and packing, as well as other board materials, plaster boards and related jointing materials.

Q. And then in 1943 the Illinois Labor Bulletin. Was there publications concerning gaskets as being a concern during that time period?

A. Yes. The Labor Board emphasized gaskets as one of the principal asbestos-containing products with the need to apply wet methods and dust suppression, the same techniques that were talked about this morning.

Q. And, sir, Richard Doll, did he, in 1955, publish

an article concerning asbestos?
A. Yes. Richard Doll looked at the textile workers again. This time not in terms of asbestosis, the scarring disease, but in terms of lung cancer, and found that the textile workers were at tenfold increased risk for developing lung cancer, highly statistically significant. And he really definitively established the association between asbestos in the textile industry, Chrysotile asbestos, and development of lung cancer.
Q. And that was all Chrysotile that Dr. Doll was dealing with?
A. Yes. It was the textile industry, which was Chrysotile.
Q. Okay. And then in 1958, did the AIHA -- we've heard a lot about the AIHA. They published things over the years, the AIHA, correct?
A. They certainly publish recommendations in terms of exposure and worker protection.
Q. And this Industrial Hygiene Organization, did they talk about gaskets and packings being a source of asbestos exposure?
A. Yes. Among other materials, the same types of materials we've talked about, and the concern for asbestosis and lung cancer.
Q. Okay. And then Dr. Wagner in 1960. How is that

relevant to understanding asbestos and asbestos cancers?
A. Certainly Wagner added knowledge regarding Mesothelioma and concern for Mesothelioma and asbestos developed in the late 1940s. Cases were observed in the Canadian Chrysotile mines; two cases reported in 1952. And Wagner followed up with a much larger study in the northwest cape of South Africa identifying over 30 cases of Mesothelioma among crocidolite miners, blue asbestos miners, in the Cape and, again, added significantly to the knowledge, really establishing definitively that asbestos was a cause of Mesothelioma.
Q. So even prior to Dr. Wagner, there were reports of Chrysotile workers, or Chrysotile miners, getting Mesotheliomas in the literature?
A. Yes. Cartier published that in 1952. Two of the Quebec miners and millers were Chrysotile-exposed.
Q. You and I haven't focused much on it because we've been focusing in on gaskets and packing. But all along this timeline from even before 1930 in Merewether and Price, there are other articles that deal with what was known and knowable about asbestos and whether it causes asbestosis, lung cancer and Mesothelioma that you and I aren't talking about. Correct?
A. Oh, certainly. I mean, through the decades there are hundreds. And if you go to the current time, there's

thousands of articles that address the growing medical knowledge regarding asbestos-related disease and refining that knowledge.

Q. And so you and I are focusing mostly on gaskets and packing and what was known about those, except for the Doll case or the Doll paper and the Wagner paper. Correct?

A. Yes. Those would be other areas.

Q. Okay. And then Dr. Hueper. In 1964, did he publish at the Selikoff conference and give a presentation about the different types of products that had potential for asbestos disease?

A. Yes. That was discussed in the 1964 Selikoff conference. It was published in 1965. But Hueper re-emphasized the concern for asbestos-related products, among which included gaskets and packing and other materials such as insulation, friction products, asbestos cement and other materials. But, this time not just in terms of asbestosis and lung cancer but also Mesothelioma.

Q. Would it be fair to characterize that 1964 conference as only being about either crocidolite or just about insulators?

A. Not at all. The 1964 conference was really organized by Selikoff to synthesize the knowledge of

asbestos at that time, given the growing volume of research. And it really related to asbestos broadly in terms of all of the major commercial fibers and a broad range of materials.

Q. Now, Dr. Brodkin, I want to talk to you about your methodology at this case, and particularly in dealing with the questions of whether Chrysotile asbestos causes Mesothelioma and whether Chrysotile asbestos in gaskets or packing caused Mesothelioma. Okay?

A. Certainly.

Q. And I have methodology. Have you gone through a methodology in trying to answer those particular questions as an Occupational Medicine doctor?

A. Yes. There are three major methods that I use in my practice and that physicians in Occupational and Environmental Medicine would typically use in addressing the question of causation, whether a material like asbestos or Chrysotile in particular caused a disease like Mesothelioma. And those three methodologies, which are not mutually exclusive -- in fact, I would call them complimentary -- would include the occupational and environmental history, which is a practice fairly unique to our field in terms of taking a comprehensive occupational history. Secondly, the Helsinki Consensus criteria which establishes how or what criteria are

necessary to establish an asbestos-related disease. And thirdly, the Bradford-Hill criteria for causation, some of which was talked about earlier.

Q. And I guess my spell check didn't work very well. The environmental history and occupational history, is that something that's important?

A. It is. It's really integral to my field of occupational medicine. One can't evaluate it unless one's gone through a comprehensive occupational history.

Q. In regards to gaskets and packing exposures, say in people who are pipefitters or people who are machinist's mates on board ships or any job where they would do gasket-type work. Have you looked at that and seen those type of occupational histories in the past?

A. I have. As I said, I've interviewed hundreds and hundreds of workers over the years that have participated in those activities, particularly in shipyard settings, but also land-based settings.

Q. And this methodology that you and I are going to discuss, has this been something that's been published in books like your own?

A. Yes. Certainly, the process of identifying risk hazards and exposures through the occupational history is a methodology that's integral to the field of Occupational Medicine. We Certainly published it in our

textbook. That section was published by Dr. Cullen and Dr. Rosenstock, leaders in the field, on how one identifies exposures. So, that really underlies the occupational history. And to identify an exposure, one needs, in terms of asbestos, a documented source of asbestos and then an activity disrupting that source.

Q. So this methodology that you and I are going to use has been published in textbooks which are the types of books that deal with Occupational Medicine and are generally accepted in that field?

A. Certainly. Including our own.

Q. Okay. Now we were talking about occupational history. What as an Occupational Medicine doctor and, using this methodology that's been used and published, what are you looking for when you're looking at an occupational history?

A. My process and the process of physicians in my field is to identify exposures that place individuals at risk for disease. And as I indicated, one starts with the source. There has to be a documented source of asbestos-containing material. That isn't sufficient -- just asbestos and a source isn't going to create exposure. There has to be an activity that disrupts that source that generates airborne fibers that can become respirable or breathable. And to do that, there has to

be a sufficient concentration to overcome the body's defenses.

The body has defenses to cough particles that are inhaled, or to produce mucus that removes particles or fibers. So, concentrations have to be sufficient to overcome that to enter the respiratory tract, that's called bio-availability, and then enter the tissues and add to the body's burden of asbestos.

Q. Now, this -- you weren't here for this but Dr. Longo and others have done gasket fabrication studies and numbers. I showed these to you this morning; correct?

A. Yes, that's correct.

Q. And there's power wire brushing. These are Dr. Longo's numbers again. Are you familiar with these type of numbers?

A. Certainly. I've read Dr. Longo's reports and others, such as Dr. Millette and other reports, regarding ranges of exposure and, certainly, these ranges are within the ranges I've seen.

Q. When we talk about ranges of exposure. Even if we took -- if we set aside Dr. Longo's reports and we set aside Dr. Boelter's reports, are there publications in the peer reviewed literature that talk about gasket exposures that, as an Occupational Medicine doctor, you

can look at and decide whether there's the availability for people to be exposed to asbestos-containing gaskets doing the typical work practices of changing a gasket, grinding, and those type of things?

A. Yes. These studies are essential because they really describe the airborne exposures when that activity is performed. So that relates to the occupational history. If a worker is hand grinding a gasket, that generates a certain airborne exposure. If they're power wire brushing a gasket, it's going to generate a higher exposure. So it's really these studies that inform me about the airborne exposures.

Q. And these studies that inform you, it's a wide range of sources that you've looked at. I mean, it's not just one or two data points?

A. That's true. I don't think one can just pick out a study and say this is Exposure X. One's talking about a range of exposures from relatively lower exposures to relatively higher exposures, and one wants to look at the whole range.

Q. Now you talked about -- so we talked about source, and that would be -- you've got to put asbestos in the gasket or packing; is that correct?

A. Yes. Typically, during historic periods in hot applications or high pressure applications, those would

be asbestos-containing gaskets.
Q. And in the past, have you seen cases involving Garlock gaskets?
A. I have in the past. Yes.
Q. And those generally had a high level of asbestos content in them?
A. If they were for hot applications during those historic times, yes.
Q. And what levels of content would those be?
A. Typically, in the 70 to 90 percent range. Some may be lower, some may be higher, but typically in that range.
Q. Now, what type of activities are you looking for as an -- as an Occupational Medicine doctor who deals with this issue of whether individuals are at an increased risk for diseases like Mesothelioma?
A. Exposure to gaskets from the occupational history really relate to activities that would disrupt the fibers from the encapsulation, and that would be activities ranging from hand activities with scraping during installation with ball peen hammering, scissor cutting, to fabricate gaskets. And then during removal, either hand scraping or wire brushing, or then at higher levels power brushing, either with pneumatics or other power tools.

Q. And in fact, when a gasket is just sitting there in place, or maybe just -- we had one here, even though we have to have it, you know, in a special bag. If it's just sitting there and not being manipulated, is that a problem?
A. I would not identify that as an exposure gasket. A gasket in the form of a pre-formed gasket? There's no activity there generating airborne fibers.
Q. And so what has to happen for it to be bio-available?
A. That material has to be disrupted. Either during installation -- if it has to be fit, cut, it certainly could release fibers. Or if it's removed or degraded, that disrupts the material and generates very significant airborne fiber levels.
Q. And I know you haven't been here, but there's been a lot of discussion of Dr. Selikoff and that 1978 statements. Is there some distinction there that they also draw?
A. Well, I noted this morning there was discussion of the Harries table where it's indicated that the form of the gasket used in shipyards is not considered hazardous, and I think that really gets to the source. Just the source sitting there is not an exposure per se, but the Selikoff chapter really emphasizes activities not

specific to gaskets but to all materials that if there are activities disrupting an asbestos-containing material, that results in an exposure of concern to workers. So it really distinguishes the form from the manner in which it's used or the activity, and that's an important distinction.

Q. And we've talked a lot about exposures of that background. Would you -- do you have an opinion whether individuals that are doing this type of work, whether they're punching out a gasket, whether they're grinding it with a hand wire brush or they're taking an electric grinder, would those type of work activities expose individuals to asbestos if it's in that gasket above ambient or background levels?

A. Well I would probably use the term "ambient." Because "background" in Occupational Medicine can also mean bystanders. If the worker next door is doing it, that can be a background exposure. So I would distinguish that from ambient, which is in the general air we breathe. These are exposures that I would characterize as much higher -- high level compared to low ambient levels. In fact, they would be in the range of 60,000 to 30 million times higher than ambient levels that have been reported.

Q. And that's not just using Dr. Longo's numbers;

that's using your comprehensive review of the scientific literature?

A. That's true. Yes.

Q. Now, you talked about the body burden. Can you explain what you mean by that?

A. "Body burden" is the concentration of the toxicant, in this case asbestos, within the tissues. And in the case of asbestos, these would be fibers that were inhaled, that overcame the body's defenses and then entered the lung and, as was discussed by Dr. Brody, can then translocate to the pleura membranes and other areas of the body.

Q. And how does dose-response play into all of this?

A. Dose-response is an important part of the information derived from the occupational history because a disease like Mesothelioma is a dose-response disease. The greater the dose, the greater the risk for disease. So we've talked about the levels, the ranges related to work with gaskets and packing that can be, you know, less than a fiber per cc up to the tens of fibers per cc.

If one compares that to the dose necessary to develop Mesothelioma, and a number of important studies that are cited on this slide from large Mesothelioma registries indicate that that's in the range of about .07 fibers per cc to .99 fibers per cc. At that dose range,

cumulative dose range, there's a three to eight fold increased risk for Mesothelioma, highly statistically significant. So if an individual from the occupational history is performing activities in the one to tens of fiber per cc range on a career basis, certainly they're going to be well above the range established for the dose-response for Mesothelioma.

Q. Now, what are these registry studies? What are we looking at?

A. We're looking at the French and German registries. Both have over 400 Mesothelioma cases. Both did a very comprehensive job, exposure matrix and reconstruction of the dose of individuals that became registered and are designed to look at Mesothelioma risk and a wide range of exposures from the fiber per cc to less than fiber per cc range.

Q. Are these registry studies, are they able to tease out whether an individual was only exposed to Chrysotile or whether they were exposed to some amosite or they were exposed to both fibers?

A. No. These are, essentially, national registries. And there are individuals that certainly would have been exposed to all fiber types in those registries. So, they don't -- they're not designed to uniquely look at a specific fiber type.

Q. And in that type of study, could you actually design a registry study that could do that?
A. You couldn't. And in the real world we're not talking about a Chrysotile world, an amosite world and a crocidolite world. These are fibers that were commercially used. While Chrysotile was the predominant fiber, with about 95 percent use in North America and Europe, there were certainly Amphiboles used as well. So when you're looking at Mesothelioma cases on a national basis, you can't separate out the individual fiber types.
Q. Now we talked about the Helsinki criteria as being one of the things that you use or one of the methodologies that you use to come to your conclusions. What is this Helsinki criteria?
A. The Helsinki criteria is a consensus report. It was published in 1997. The meeting took place in Helsinki but it involved an international group of imminent researchers in asbestos-related disease in my field of Occupational Medicine, as well as pulmonary medicine and pathology, that developed criteria that, by consensus, were felt to be necessary to diagnose asbestos-related diseases which would include asbestosis, the scarring disease, lung cancer; pleura plaques, scarring in the lining of the lung; as well as Mesothelioma.

Q. And we have a few cutouts. But what they did was they had 19 different individuals from countries who were not producing asbestos and they all gathered and had collectively done over 1,000 articles on asbestos. So these aren't folks that aren't aware of the world's literature; correct?

A. These participants were selected for their experience with asbestos-related disease. And certainly, all of them had made important contributions.

Q. And this Helsinki criteria or consensus document, do they talk about attribution for Mesothelioma and other asbestos-related disease, how you do that?

A. Yes. They talk about the criteria necessary to attribute a case of Mesothelioma to asbestos and what's necessary.

Q. All right. And I have up there, "A history of significant occupational domestic or environmental exposure to asbestos will suffice." Did they talk about what they meant by a history of significant occupational exposure?

A. This really gets to the first method I used, the occupational and environmental history. You have to take a history and get that exposure information, and that's essential to the diagnosis. They're really speaking to that process.

Q. And did they talk about how much history you need to attribute a Mesothelioma to asbestos?
A. Well, given the dose-response from Mesothelioma, they indicated that even at the lower levels, brief or low level exposures should be considered sufficient to attribute a Mesothelioma to an occupational setting.
Q. Now you would agree with me, doctor, that at that time this question about whether Chrysotile asbestos causes Mesothelioma had been discussed in the world's literature; correct?
A. Oh, certainly for many years.
Q. And would it be safe to assume that 19 individuals who's written over 1,000 articles on asbestos, they would have an understanding of that debate?
A. Certainly. And certainly fiber types are discussed within that document.
Q. And even though it discussed fiber types, they still say in this consensus document that occupational history of brief or low level exposure should be considered sufficient for Mesothelioma to be designated as occupationally related. Is that something you agree with?
A. It is. And specifically, there's no discussion of fiber type here. It's really asbestos from the occupational history. I do agree with that. I certainly

use the Helsinki criteria to diagnose asbestos-related disease.

Q. And they don't say in the document that massive exposure to Amphiboles or massive exposure to Chrysotile is required. It just says brief or low levels.

A. That's correct. Yes.

Q. Now, are you aware of other scientific agencies that have dealt with this brief or low level exposures to asbestos?

A. Certainly. Yes.

Q. Is there a consensus view in the world literature whether low levels of exposure to asbestos, no matter what the fiber type is -- has there become a consensus view on that issue, whether it causes Mesothelioma or not?

A. There certainly is. I mean, it's recognized that Mesothelioma is a dose-response disease. The greater the dose, the greater the risk. But there is a broad dose response for Mesothelioma that includes low level exposures. So, certainly that's been recognized by all the major governmental agencies that address public health, including NIOSH and OSHA, and the International Agency for Research on Cancer, ATSDR, EPA. None of them really distinguish between a necessary high dose versus any other dose or a fiber type.

Q. And we talked with Mr. Templin about the fact that at the Selikoff conference they talked about there not being a threshold. Is that what we're talking about here, that there's no threshold for Mesothelioma in relation to asbestos exposure?

A. The scientific literature has not identified a specific threshold below which we can say an individual is safe; they will not develop Mesothelioma above which they are at risk. The French and German registries, which I spoke to, give specific doses at which we know there's increased risk, but they're not able to give a specific threshold dose.

Q. Now, this no threshold that there is no risk below which. That's where we sit today, even though there's been, I don't know, thousands of articles written on asbestos. Correct?

A. That's the status of the science today that we know certain doses where there are increased risk. I spoke to the .07 to .99 fiber per cc. We know with those doses there is increased risk, but we don't know that there is a threshold. It's possible there is one, but the studies to date haven't identified one.

Q. And Dr. Brodkin, as an Occupational Medicine doctor, are you aware of any substance that has been studied throughout the world's literature from the 1930s

on more than asbestos?
A. Well, speaking from an Occupational Medicine perspective, I would say that there probably isn't a substance that's been studied in more broadly and more in depth than asbestos. Certainly there are others that have been studied extensively, but asbestos has been researched for many decades now.
Q. Now we were talking about the Helsinki criteria. Other than the Helsinki criteria, you also referred to the Sir Bradford-Hill criteria which, I think, Dr. Brody talked about briefly. Is that something you're familiar with?
A. Yes. I didn't finish my answer on Helsinki, though, in terms of the criteria necessary to diagnose. I don't know if you want to go over that.
Q. Well, let me just -- in the context of this particular case, have you seen individuals who've worked with gaskets and packing that get Mesothelioma that meet those Helsinki criteria?
A. Yes. Because they have a defined occupational exposure with an activity-generating airborne fibers. But in addition, there has to be pathologic evidence. They have to have a biopsy that proves they have Mesothelioma. They have to have sufficient latency of at least ten years, often more, to develop the disease. And

one needs to go through a differential diagnostic process and make sure there are no other rare risk factors for Mesothelioma. But after one has gone through each of those steps of the Helsinki criteria, one can then establish an asbestos-related Mesothelioma.

Q. Okay. Now, the Sir Bradford-Hill, that's the next part of your methodology in looking at this particular issue of Chrysotile asbestos and then gaskets and packing; correct?

A. Yes.

Q. And I have up there the article -- I guess it was a speech given in 1965. And basically, he outlines different things that you need to look at. Can you explain to us what is the relevance of the Sir Bradford-Hill criteria and how you apply that in this issue of Chrysotile asbestos?

A. Yes. It's one of the important methodologies of causation. It's been discussed earlier today, as well. But this really speaks to the body of scientific evidence that's necessary to establish causation, and that gathers evidence from a number of areas. The first would be epidemiology in terms of characterizing a strength of association, as well as looking at consistency of the data and the studies and understanding dose-response. But it also considers latency, which I talked about

earlier. The temporal association between exposure and disease has to fit in terms of a recognized pattern. There has to be plausibility, which we talked about, which is really evidence for a biologic mechanism. There has to be experimental evidence, and certainly Dr. Brody has addressed that.

In addition, with asbestos, there are issues of specificity because asbestos is so specific in its association with Mesothelioma as well as coherence and analogy. Those aspects of evidence should be considered before determining whether an association is causal.

Q. And when we're talking about this association versus causal. Even though we have, I guess it's nine criteria that Dr. Hill talked about, is any one of those nine criteria, based on what he discussed, somehow heavily weighted one way versus the other?

A. I would say not. Sir Bradford-Hill indicated there really is not one single piece of the puzzle that tells you about causation. There's a body of evidence that collectively should inform you about causation and allow you to make that judgment. Certainly epidemiology is an important aspect, but toxicology, in terms of animal and biologic mechanisms, is extremely important as well.

Q. So let's look at the strength of association. You

and I are going to talk about certain articles. But there are other articles on this question of Chrysotile and Chrysotile predominantly exposed cohorts that we don't have listed up there; correct?

A. That's true. There are numerous studies that have addressed disease risk in Chrysotile exposed cohorts, probably in the range of 25 or more, but these certainly are important ones that have informed my opinion.

Q. Okay. And can you explain to us why these particular studies are important?

A. Well, these studies certainly address Chrysotile. They take place in areas where Chrysotile was the predominant exposure in its use with minimal contamination. So it allows one to look at the effect of Chrysotile as a predominant exposure. And in the case of the bottom one, the Vianna and Polan, while not exclusively Chrysotile, some uniquely Chrysotile settings.

Q. And so when we're dealing with the strength of association we have the different risks up there. What does that mean and how does that relate to this issue?

A. As I'm informed has been discussed with epidemiology. Certainly, one looks at strength of association as disease occurrence in an exposed group compared to an unexposed group. These studies are

designed to look at that. And when one sees a doubling of disease occurrence in the exposed compared to the unexposed, that yields a relative risk ratio of twofold or a hundred percent increase in risk. Now in the case of these studies, the risks increase 400, to over 3,000 percent. So, fourfold to 33 fold increase. These are very high magnitudes of association. They're very robust in establishing a very strong association and certainly would indicate that a high majority of cases in these groups would be attributable to asbestos.

Q. Now, Dr. Brodkin, first of all, part of your training in Occupational Medicine involves epidemiology; correct?

A. Yes. In the fellowship training for Occupational Medicine, physicians are required to get a master's in Public Health to understand the principles of epidemiology, bio-statistics and, in my field, toxicology and industrial hygiene. So, yes, there is extensive training.

Q. And I think I shortchanged you trying to get you on quickly. You do have a master's in Public Health, besides having your degree as a medical physician?

A. Yes. I did my fellowship at the University of Washington and concurrently, as is required, obtained my master's in Public Health.

Q. Now when we're looking at these issues, we're only looking at the ones that are 400 to, I think you said, over 3,000 times risk?
A. Correct.
THE COURT: No. Percent.
BY MR. FROST:
Q. Percent. I'm sorry.
A. Yes. 3,300 percent.
Q. Now there are other studies that either show lower risks or show no risk.
A. There are. And there are other studies that don't look at risk in the same way. They look at rates of disease. There are numerous studies that do that, and that gives you the rate of disease that you can compare with other populations. It doesn't, within the study, compare it to an unexposed group, but certainly you can use that to look at the disease risk. And there are studies, the South Carolina textile workers, the studies out of Zimbabwe, that generate rates of disease that also show high rates among Chrysotile-exposed workers.
Q. And those don't -- basically, they don't calculate a relative risk. They just give rates of disease; is that right?
A. True. And the same for Quebec miners and millers. They have a very high rate of Mesothelioma, but those

studies don't generate relative risk metrics.

Q. Would it be fair to say that the only place we see individuals at an increased risk of Mesothelioma involving Chrysotile only appears in people who are miners and millers of Chrysotile?

A. No, that's not true. Certainly, miners and millers are a group at risk. And those workers in Canada, Zimbabwe, South Africa have certainly been reported on and provide important information. And also the study I've cited in Sverdlvosk, Russia are mining areas.

MR. SCHACHTER: Excuse me, Your Honor, I don't believe that study was disclosed in his report. If they're going to talk about it, I would like to get a copy. I object to it if it's not in his report. The Wang article is not in his report, unless I've overlooked it. But if they're talking about new material --

THE COURT: If they're not in the report, we'll not talk about them.

MR. FROST: To be honest Your Honor, there were a lot of studies in the report. And I'll take his --

THE WITNESS: My understanding is they are in the report.

MR. SCHACHTER: What reference number are they? Because we tried to get a copy of them. I don't mean to

address the witness.

MR. FROST: That's fine. Dr. Brodkin -- grab your list, Dr. Brodkin.

THE WITNESS: Excuse me?

MR. FROST: Grab your list.

THE WITNESS: Wang and Lin is reference 807 in the first report.

MR. SCHACHTER: Okay. I was wrong.

MR. FROST: I'm sure it won't be the last time. Is there another?

MR. SCHACHTER: Yeah. I didn't see the first one.

MR. FROST: Is it okay to continue?

MR. SCHACHTER: I would like a copy of Becklake.

THE COURT: All right. Let's go ahead.

THE WITNESS: Yeah. I'll -- on a break or whatever, I can look for that.

BY MR. FROST:

Q. Okay. Great. So Dr. Brodkin, we're still dealing with the strength of association. Is there anything else -- when we're dealing with that issue on the Bradford-Hill criteria, we're sort of hitting the highlights. Anything else that's important on this issue of Chrysotile asbestos?

A. Well I think the other point is that there is also consistency. We're seeing consistent magnitude of risk

that's highly positive among the studies. So, that's the consistency criteria also for the Bradford-Hill criteria.

Q. Now the Court's already seen this, I think, a couple of times. But you're aware that some studies -- there is some allegation somewhere that there might potentially might have been some Amphiboles that might have been present in small quantities in certain areas. If you look at it one date and one time period, there might have been some crocidolite or something used. Does that invalidate those Chrysotile cohorts?

A. In my opinion, not at all. One is looking at Chrysotile as a predominant material which in Occupational Medicine is important because you're looking at the effect of an exposure, which is predominantly Chrysotile. So one goes to areas where predominantly Chrysotile is used.

I guess there are two answers to that question. One is a mineralogic question. If you look under a microscope and a mineralogist looks at it, can they find impurities that would include Amphibole? And the answer is, in many of these cohorts, yes. Such as in the Canadian Thetford Mines, you will find up to one percent Tremolite. In others, like Chunking, China, very low levels, within the 001 percent; a very small fraction of one percent in terms of contamination. But, one can find

that mineralogically.

From an Occupational Medicine perspective, I don't find that terribly important because I'm really looking at the dominant exposure, and the world isn't pure exposure. I mean whether one is looking at the mineral in the earth or the end product. If one's looking at gaskets and packing, those are predominant Chrysotile exposures but they have contaminants in them, too. In many cases, that doesn't bother me. I mean, I'm looking at a Chrysotile of predominant exposure. I don't make that distinction as an Occupational Medicine physician, although I understand that mineralogists certainly can.

Q. And in fact, when we go back to the -- I always get it wrong -- Balangero cohort. Those folks have been studied extensively; correct?

A. They have. In Balangero, while they didn't find any Tremolite in the ore itself, they did find a fraction of the a percent of Balangeroite, which is another mineral that has been discussed whether it could cause health effects or not.

Q. As we sit here today, are there peer reviewed published articles that say that the Balangeroite, I guess would be the right way to say, it causes Mesothelioma in that cohort?

A. Well that question has been raised. That

Balangeroite has been studied by Italian investigators. Favero-Longo, in the Journal of Toxicology and Environmental Health, 2009, published the results of their research. They felt it was unlikely that that would contribute to be Mesothelioma based on its low bio-persistence ability to persist in the tissues. They felt that that was likely not contributing significantly to Mesothelioma. But there are -- it's certainly been an area that's been under investigation.

Q. Now, the next area is temporal association. What do we mean by that when we're talking about this Bradford-Hill criteria?

A. That really is very similar in the Helsinki criteria to latency. That predictable time between exposure and development of disease and, certainly, among all the different fiber types that latency is very similar. It generally requires at least ten years but, on average, may be in the range of 35 years and can be up to more than seven decades in some cases. And that would be true whether it's Chrysotile or the Amphibole fibers.

Q. Is that something you've seen not only in your CARET study but in your practice of individuals, such as pipefitters, machinist's mates, people like that?

A. Yes. The mean latency period in the CARET study was 35 years, which is pretty typical of other studies as

well.

Q. Now, the next area is dose-response. What are we talking about when we deal with dose-response?

A. Dose-response. We've -- in terms of the Bradford-Hill criteria it really indicates there evidence that would increasing dose. There's increasing risk for disease. And we talked about that for asbestos in general. But there's certainly evidence for Chrysotile as well as a specific mineral. First of all, there is evidence, as Dr. Brody indicated, that Chrysotile preferentially is able to reach the pleura tissues and concentrate in the pleura issues, which is the target organ for development of Mesothelioma. So that's important evidence.

Dose-response at the tissue level -- and certainly a number of studies have looked at lung burden of Chrysotile as well and found a strong correlation. Rogers and Lee found an eightfold increased risk for Mesothelioma with increasing lung burden of Chrysotile. So I think there is significant tissue burden evidence that, with increasing dose of Chrysotile, there's an increasing risk for disease.

Q. And dealing with this issue. Dr. Suzuki published on this, whether short fibers translocate from the lungs to the pleura tissue. And what do we mean by

"translocate?"
A. Translocation really refers to what Dr. Brody explained is that the kinetics or movement of the mineral fiber within the body. So, the ability for an inhaled fiber to get from the lung out to the pleura via the lymphatic channels.
Q. Now we heard a lot of discussion from the defense experts about, well, amosite collects in the lung tissue. Amosite? Look in the lung tissue. Look in the lung tissue. Why is that important to look in that lung tissue when we're dealing with the issue of causation of Mesothelioma?
A. Well, certainly, it can be useful to look in the lung tissue. And for Amphiboles, that's a fairly simple way to look for it because it's very persistent in the lung tissue. In the case of Chrysotile, it's not a sensitive way. The half-life for Chrysotile in the lung is about three months. If you look a year later, most of the Chrysotile will have broken up and translocated and moved to other areas of the body.
So, looking at lung tissue is not going to give you a sensitive metric for distant exposures to Chrysotile in the same way that looking at pleura concentrations would. Lung burdens are used because they can be done in laboratories and are available, but they

don't reflect old Chrysotile exposure very sensitively.
Q. And in fact, if I have an individual who was exposed to Chrysotile, say, 30 years ago, would we expect to see that -- those Chrysotile fibers in their lung if we looked at their lung using fiber tissue digestion?
A. In most cases, you will not see the Chrysotile after that length of time. The kinetics of Chrysotile again such that it will not persist in the lung for those periods.
Q. Have there been studies that have looked in the other places? Because the asbestos has got to go somewhere; right?
A. Well, certainly, it is a property of Chrysotile to translocate to other tissues.
Q. And have there been studies looking at how it gets from the lung to the other parts of the body?
A. Yes. Pathologists have looked at tissue concentrations. I mean, we've talked at one in the pleura tissue. But Dodson and Hammar and other investigators have looked at other serosal membranes, pleura-like membranes, in the abdomen, distant from the lung, and certainly one can find Chrysotile there. But in the pleura, Suzuki identified about the 30-fold increased risk -- 30-fold increased concentration of the Chrysotile compared to the Amphibole fibers.

Q. So what Dr. Suzuki found is that short fiber Chrysotile preferentially clears to the pleura the site of the Mesothelioma tumor.
A. Yes. Relative to Amphibole fibers, that's true.
Q. Now, we've talked primarily in this trial about pleural Mesothelioma. But are there other forms of Mesothelioma?
A. Yes. Any of the serosal membranes can develop Mesothelioma. So that thin cellophane-like membrane, which is the same as the pleura lining but in the abdominal cavity, can be the area of Mesothelioma. That's called a peritoneal Mesothelioma. The serosal membrane surrounding the testicle, the tunica vaginalis, can be a source of Mesothelioma. So those distant sites have been described as well.
Q. What about the serosal membrane around the heart?
A. It can as well. That's a rarer entity. Usually, pericardial involvement is from direct spread from the pleura into the lining of the heart, that accounts for a great majority of cases. But, at times, you can get a primary pericardial Mesothelioma.
Q. And even though there's -- these are in different parts of the body, what's the common thread that we have in causation with those diseases, those Mesotheliomas of the serosal membrane?

A. The common feature is asbestos exposure. And certainly, the Bradford-Hill criteria can be looked at in terms of these other serosal membranes. But as the Helsinki criteria emphasizes, any serosal membrane can be the result of asbestos exposure in terms of Mesothelioma. So that distinction is not made.

Q. Now, once asbestos gets into the lung and the Mesothelioma process begins, can you explain to us what we're looking at and how that process develops in a human being?

A. This is an autopsy slide of an individual with a right side of Mesothelioma, the right side of the chest, that being on the left side of the slide. This is both lungs. But the normal appearing left lung on the right side of the slide. If you look at the exterior of it, you're not aware of the pleura. It's a very thin cellophane-like membrane. But the right lung on the left side of the slide, you see a very thick white tissue lining around the entire lung. That is spreading within the fissures as well. That's called a tumor rind. That's how Mesothelioma spreads, along the pleura serosal membranes, and that's what you're saying. That thickness of tissue is the tumor of the cancer.

Q. So Dr. Brodkin, I don't want to have you come down. But what we're talking about is the lining of the

lung all the way around here, and then this is the tumor?
A. That's correct. Yes.
Q. And then what are we seeing here?
A. This is a cross-section, various sections of lung. But this really demonstrates the progress or progression of the tumor. It goes from a tissue rind surrounding the entire lung to the formation of thick nodules and eventually the obliteration of the normal lung tissue, and that's how death occurs.
Q. And so at the top, what are we seeing there?
A. Again, this would be a rind, an encasement, of tumor around the entire lung.
Q. So that white area at the top is the beginning of the tumor? The rind?
A. And it goes below. It completely encases the lung.
Q. And then this is the continuing development. And then this black area, that's the lung?
A. That's the lung. But here in this section you're seeing the tumor nodules, those masses, as the tumor becomes thicker.
Q. And then at the bottom, what are we seeing there?
A. This is an area where the nodules have become so large that they've obliterated the normal lung and you don't see it.

Q. And so, really, what we're left with is what happens at the end state of Mesothelioma?
A. One's basically looking at a strangulation of the lung. Death is from respiratory insufficiency.
Q. And is that the common end state for Mesothelioma victims?
A. For pleural Mesotheliomas, that's true.
Q. Now doctor, we've talked about -- we're still talking about applying the Sir Bradford-Hill criteria. We're talking about dose-response. Has there been studies that have dealt with dose-response and Chrysotile?
A. There have been. It's harder to do those studies because you have to go in areas where only one fiber type is used. Certainly, the Madkour, in Egypt, looked at a asbestos cement process that used Chrysotile that had 80 or so Mesotheliomas and does provide evidence of a dose-response. The median dose of the Mesothelioma cases was seven fiber per cc years, half of the exposures of less than that.
Q. What they did is they took a plant that, in their article, they describe as an asbestos manufacturing plant using Chrysotile asbestos, and they looked at individuals who either were working in the plant or live around the plant to see how many Mesothelioma's they had?

A. Correct.
Q. And what did they find when they started to look at that issue?
A. They found about four Mesothelioma cases among the plant workers. The majority of them -- over 80 of them, I believe, were in the environment around the plant. And certainly, they looked at the occurrence of disease as individuals lived further and further away from the plant to see if there was evidence of dose-response.
Q. And so the point in the middle here is where the plant is. And then what they did was they tried to look and point out and find as you went farther and away from the plant whether you had Mesotheliomas?
A. Correct.
Q. And these are the results. I don't know if we really need to go through that. But what's the importance of that particular study when we're talking about dose-response?
A. Well, certainly, they found a higher occurrence of disease as you got closer to the plant. And that occurrence got less as you went further away, and that's certainly evidence of dose-response. Certainly, the median fiber cc year exposure for the individuals at 75 per cc years is within a broad range that's consistent with other studies. It's not exactly the same, but it's

certainly gives more specific information.
Q. And have there been other folk that is have had indirect exposure to Chrysotile that show these same types of increased risk for Mesothelioma?
A. Yes. In the Balangero mines in Italy, of the 27 Mesothelioma cases, two of those were in individuals with what we call indirect exposure. They didn't directly work with the mining material. One laundered the clothes of one of the workers and one collected leaves in the area but didn't work directly with the material. So that's an example of indirect exposure and is evidence that exposure is occurring at lower doses of Chrysotile.
And similarly, in Canada, looking at the experience of Mesothelioma in women who did not work at the mines but lived in proximity to the mines. About a sevenfold or 700 percent increased risk in Mesothelioma was identified by Camus.
Q. Now we've gone through dose-response. How about consistency? What have we seen in that when we apply the Sir Bradford-Hill?
A. Consistency, I spoke to when we talked about the epidemiologic data. All of those studies show a fairly high magnitude of risk, and consistently show an increased magnitude of risk. We're not seeing studies that show reduced risk. They're all in the same

direction.
Q. And what about biological mechanism? How do we relate that to this issue?
A. Dr. Brody talked about plausibility. And certainly, that body of work informs my opinion as an Occupational Medicine physician that through toxicology each of the fiber types is potent in causing Mesothelioma. So that's another body of evidence I look at that's independent of the epidemiologic evidence.
Q. I think that's a good point. We've talked about a lot of government agencies and different studies and things, but is Chrysotile asbestos listed as a known human carcinogen?
A. It is. The agency that's charged with doing that the physicians in my field utilize is the International Agency for Research on Cancer, and they've designated asbestos at each of the major commercial fiber types Group 1A, Known Human Carcinogens. So that is each of the fibers is treated in that fashion.
Q. Now the other one is animal studies, and Dr. Brody's talked a little bit about that. But have you also reviewed those type of studies?
A. Yes. And I think the biologic mechanism and the animal studies are very interrelated.
Q. And then the next one is specificity. How does

that -- what have we seen in the world's literature concerning that?

A. Well, specificity is a criteria that's a met for each of the fiber types because Mesothelioma is uniquely associated with asbestos exposure. There are very few other causes of Mesothelioma. Rarely, therapeutic radiation will cause it. Rarely, a chronic injury like a recurrent collapse of the lung can be associated with a Mesothelioma. But asbestos is -- well, Mesothelioma we really call a signal tumor for asbestos exposure because there's such a unique relationship. And that's the same for Chrysotile as it is for the Amphiboles.

Q. And when we talk about a signal tumor. If an individual goes to their doctor and they've sent off their samples to a pathologist and the pathologist has looked at that and done the immunohistochemistry chemistry and decided that this is in fact a Mesothelioma, what is one of the first questions the doctor asks the patient concerning their past exposures?

A. It should trigger an occupational history. Now many physicians outside the field of Occupational Medicine don't take that comprehensive history, but it should trigger the question, was there asbestos exposure?

Q. And in fact, have you seen that in medical records in the cases you reviewed and the things you've been

involved with when an individual is diagnosed with Mesothelioma, one of the very first things their doctors asks them how were you exposed to asbestos?

A. That's often a discussion that takes place. Yes.

Q. Okay. And signal tumor. Besides Mesothelioma being a signal tumor, is asbestosis a signal disease?

A. Asbestosis is unique to asbestos. But the process of diagnosing asbestosis, I would say, is distinct from Mesothelioma because there are many other diseases that cause scarring of the lung. So one has to go through a significant differential diagnostic process. So, as a disease state, fibrosis of the lung should really trigger an investigation of all potential types. But that investigation if it has sufficient criteria and will lead to the diagnosis of asbestosis, and that is unique to asbestos.

Q. In fact, asbestosis, the term was coined because it was caused by asbestos?

A. That's correct. Cook described that in 1927.

Q. So even though we had Merewether and Price in the 1930s where there were reports of this type, were there reports of this type of disease prior to that?

A. Yes. I would say the first characterization of asbestosis as such, independent of other dust-related diseases, would be 1927.

Q. Now we're still talking about the Hill criteria. We're on eight and nine, and we'll put those together. Coherency and analogy. How does that play into this analysis?

A. Well I lumped those together in the case of asbestos because one can look at other asbestos-related diseases. Asbestosis pleura plaques, which are scarring in the lining of the lung, lung cancers, each of the fiber types. Chrysotile, amosite, crocidolite, they all cause each of those diseases. In fact, there's not a potency difference in causing those diseases. So by coherence and an analogy, there shouldn't -- there should be a similar behavior in terms of cancers of the pleura, Mesothelioma. And in fact, one sees that because each of the fiber types is potent in causing Mesothelioma. So it's coherent with the other known asbestos-related diseases.

Q. And there's been a lot of discussion about all the international agencies and some kind of, I guess, allegation that you really shouldn't look at these because these agencies, they're trying to be overprotective of workers. Is that something that's a valid criticism of IARC and the World Health Organization and the EPA, all these different international organizations? Is that a valid criticism?

A. I don't think it is a valid criticism. And certainly, if one looks at the literature from these agencies one sees a careful review of the scientific and medical literature just as physicians do. Physicians participate in these committees, as well as other scientists, and they look at the broad body of scientific literature before making their recommendations. So I think these are informed decisions based on the science.

Now in the case of OSHA, there may be other practical limitations for a regulatory limit that actually may be less protective to workers just because of feasibility of application. But I would not characterize these agencies as being overprotective. I think they want to get it right. I think they want to accurately assess the risk.

Q. And so do you look at what these agencies have said and done in review in formulating your opinions?

A. These agencies inform me and physicians in my field. I mean, I read the IARC recommendations and, certainly, OSHA, NIOSH, EPA, ATSDR. They inform my opinion as an occupational medical physician. I rely on them and consider them. Yes.

Q. And this International Agency for Research on Cancer. They've looked at this issue of Chrysotile. The World Health Organization, they've actually published

pamphlets and books on that and gone through all of the analysis of things we're going to hear on cross-examination and then drawn their conclusions; correct?

MR. SCHACHTER: Objection, Your Honor. He can't possibly know what I'll ask on cross-examination.

MR. FROST: I'll withdraw that question, but I'm pretty sure I know what he's going to ask.

BY MR. FROST:

Q. Dr. Brodkin, these agencies, they didn't just publish something on the Internet that says Chrysotile is bad. Have they looked at things, as scientists do, in formulating their opinions?

A. Yes. In terms of my reading of these opinions and -- particularly, the International Agency for Research on Cancer, I mean, they are the agency designated with the responsibility of establishing whether a material is carcinogenic. It has huge implications. These are major commercial fibers. They have to go through a very rigorous process. And IARC has published periodically on asbestos over many, many decades and updated their analyses.

Q. We're not talking about things that are out of date. I mean, this is ongoing. Research continues and people submit whatever they want to try to change their

mind. That's continued over the years?
A. That's true. Yes.
Q. Okay. Now when we take that Sir Bradford-Hill criteria. Has there been published articles that do exactly what you and I did where they take the Sir Bradford-Hill criteria and apply it to the Chrysotile asbestos?
A. Yes. Others have done that. I mean, here is a commentary by a Richard Lemen going through that process and reached similar conclusions.
Q. And he was one of the former directors of NIOSH and an assistant United States Surgeon General?
A. Yes, that's my understanding.
Q. In fact, Dr. Lemen is a trained epidemiologist?
A. Yes.
Q. In fact, Dr. Lemen, was he not NIOSH's epidemiologist during a lot of the asbestos years and as NIOSH was coming up with asbestos regulations?
A. My understanding is he was a NIOSH epidemiologist. Yes.
Q. And you're aware Dr. Lemen does testify for plaintiffs in asbestos cases?
A. I am aware of that. I can't really speak to his testimony but I'm aware of that.
Q. Okay. Now, other than the studies you have of

Chrysotile, if we continue on and apply those Hill criteria to gaskets and packings, is that an area you've also taken a look at?

A. Yes. Certainly.

Q. And what's important when we're doing that, when we're trying to apply the Hill criteria, to gaskets and packing?

A. Well the major consideration is that these are Chrysotile materials. And the Chrysotile in gaskets and packing is no different than the Chrysotile we've just talked about in terms of the methodologies I've used to establish that Chrysotile causes Mesothelioma. There is nothing unique about the Chrysotile in gaskets and packing that wouldn't apply to all of the Bradford-Hill criteria that we've talked about. That being said, there are specific studies that have looked at workers who utilize gaskets and packing and certainly inform my opinion about the strength of association for those materials, and there is strong evidence.

Q. And I think it's a good point you to bring up, Dr. Brodkin, this question -- and I think there were questions to Dr. Brody as well -- well, you haven't used ground up gasket material in your studies. Is there any difference between Chrysotile when it's in a gasket and Chrysotile when it's in a joint compound or in any of the

3,000 products that it may have been in over the years once it's been abraded and it gets into the breathing zone of workers?

A. Chrysotile is Chrysotile once it's been released. Now in its encapsulation form, or in whatever physical form it's in, it may differ. But what we're talking about in terms of biologic effects or health effects is the Chrysotile fiber. So once it's released, we're talking about the same material. It's the Chrysotile fiber.

Q. It's the fiber, stupid?

A. It is the fiber. That's what causes the biologic effect. Your body sees and reacts to the fiber. It doesn't see the source. The source is irrelevant at that point. It's really the body's reaction to that and the body burden that causes risk for disease.

Q. And let's say, for example, I was an individual working with a Chrysotile containing thermal insulation product and I liberated asbestos dust. Would that be any different than working with a Chrysotile containing gasket?

A. No. And certainly one would want to characterize the activity. There can be ranges of exposure. We've talked about them for gaskets from less than a fiber per cc to tens of fibers per cc. The same would be true for

insulation. We've talked about in shipyards. It can even get into the hundreds of fibers per cc during large rip-outs of ships. So one would want to know the activity from the occupational history. But there is a wide range of exposures. Someone using power tools to remove a gasket may have higher exposures than someone hand removing insulation and vice versa.

Q. Now we're talking about applying the Bradford-Hill criteria to gaskets and packing. The first portion we have is gasket manufacturing. Has there been studies of individuals who were involved in gasket manufacturing that involved Chrysotile asbestos?

A. Yes.

Q. And can you talk to us a little bit about what's been found?

A. Yes. There are a couple of studies that address this. The MacNeal-Chicago registry characterizes 32 cases of Mesothelioma. Seven of those cases, 21 percent, had occupational exposure at a Chrysotile gasket manufacturing plant or some others at a Chrysotile insulation manufacturing plant. And then looking at the cases in that area, 27, or 84 percent of them, had a close residential proximity to the manufacturing plants as well. So, that's certainly evidence of risk. And similarly, in a NIOSH study of a Chrysotile packing plant

that actually made packing textiles and friction products, there were 17 cases of Mesothelioma among somewhat over 3,000 workers. That's a rate of .5 percent. Again, strikingly, high for a disease in the general population that would occur at one in a million.

Q. And we hadn't talked about that much, but Mesothelioma is a rare disease.

A. Right. The .51 percent would be essentially one in 200 individuals, rather than one in a million.

Q. Now these folks that were in proximity to the manufacturing plant, why is that important when you're looking at these issues in the Hill criteria?

A. Well it's similar really to the Madkour study looking at individuals that may have lower levels of exposure in the environment around a source facility, and I think one sees evidence of that here.

Q. Now, gasket end users. Is it possible to design an epidemiological study that isolates those folks out?

A. I don't think there is a design that does that. Because in Occupational Medicine -- in Occupational Epidemiology, one looks at trades. One looks at groups of workers that perform a similar trade, such as pipefitters or machinists or boilermakers. You can't design a study of a pipefitter that only uses gaskets and doesn't use other materials such as insulation. The same

would be true for boilermakers or machinists. One has to look at the trade, the real world experience of those workers. And that encompasses, certainly, frequent gasket exposure, but it encompasses insulation as well. I mean insulation, gaskets and packing, were the materials that those workers were exposed to.

Q. And so we don't have any groups or cohorts of people that only used gaskets their entire life, because part of that process involves thermal insulation.

A. In terms of end users? No. The gasket manufacturing studies are more specific to gaskets. But in terms of end users? No. There's not a way to really create that artificial isolated exposure.

Q. And I know you weren't here, but even Mr. Boelter admitted when his -- when he did his study he wasn't looking just at pipe -- people doing thermal insulation but, also, there's gaskets underneath there that potentially could be exposure. You would agree with that?

A. I think that's a fair characterization.

Q. Now you said we could look at trades. Have you looked at the trades so that we can sort of tease this out a little bit?

A. Yes. There have been numerous studies that have looked at heating trades in aggregate pipefitters,

plumbers, boilermakers that would fit into this work group that frequently use gaskets. They would also have some insulation exposure as well. But certainly through their trade in it really -- their trade would use gaskets. And consistently, and we've talked about the strength of association and consistency from the Bradford-Hill criteria. One certainly sees it in this epidemiology that one is looking at four to eightfold increases in risk, whether one's looking at the North American registry which McDonald looked at, or the British Columbia registry which Teschke looked at, or the Nordic countries registry which Pukkala looked at, or the French registry which Rolland looked at. It's very similar in the high magnitude of risk.

Q. That study from 1980. That's sort of a seminal study in asbestos, isn't it?

A. It's an important study. It's a large pathological study of, I believe, over 500 cases.

Q. Would you believe someone who is an expert in asbestos and comes in and testifies about epidemiology that they would know about that study?

A. McDonald is certainly an important study in the field of asbestos related disease.

Q. We dealt with pipefitters, plumbers, boilermakers, and we've just listed some of those studies. Did you

look at some additional studies, other than those we just looked at?

A. There are many other studies that looked at these heating trades. There are a number of refinery studies. There are a number of boilermaking studies that also find high rates of disease.

Q. And then we have the last group. The last three slides talks about machinists and mechanic repairmen. What's the importance of those folks?

A. This is also a group of workers that integrally works with gaskets and packings. Machinists have to repair pumps and valves; there are inherently sealing materials in those that have to be removed and replaced. So I think it's an important trade to look at in looking at a risk for gaskets and packing, again, not independent of insulation but, importantly, in terms of frequent use with gaskets and packing. And one sees about a two to fourfold highly statistically significant increased risk whether one is looking at the British Columbia registry, the Barcelona, Cadiz registry which Agudo looked at; the Swedish registry which Malker looked at, or the German registry of Rodelsperger.

Q. Why is it important to look at these trades when we're looking at the issue of gaskets and packings and things like that and whether they cause Mesothelioma?

A. Well you want to look at the workers that came in contact with those materials in a way that would expose them. So, that could be manufacturing workers. It could be people in the environment around manufacturing plants. It could be end users of those materials. So those are the groups or trades that inform my opinion about gaskets and packing.

Q. And when we talk about exposures. You've looked at such documents like the Navy's Safety and Occupational Safety and Health Program Manual that we've talked about with some other witnesses from 2007?

A. Certainly, the safety and occupational health branch of the Navy has considered gaskets a hazard in terms of asbestos-related disease, including Mesothelioma, and recommended practices to reduce exposure.

Q. And we won't go through that. But they, at least, recognize that sheet gaskets in high temperature applications can become friable?

A. Yes.

Q. Why is that important that they can become friable?

A. Friability really relates to bio-availability. Again, asbestos is encapsulated and the source is not going to be a risk factor for exposure. But if it's

friable, meaning it can be crushed in the hands, then fibers can be released with fairly minimal physical contact and can result in exposure. So, degraded gaskets can become friable and a source of exposure and airborne fibers.

Q. And that issue is looked at by OSHA. We've gone through this with some other witnesses but -- you've seen this before, this ship and the hazardous warnings on board ships for asbestos gaskets and things like that?

A. This is specifically for ship breaking activities. OSHA recognized that in old ships you can disrupt asbestos gaskets and get exposure.

Q. And the EPA has done the same thing in regards to scrapping of old ships?

A. Right. The same thing. Removal of old gaskets that were used in the hot applications in the mechanical spaces of vessels.

Q. As an Occupational Medicine doctor, why is it important that the EPA recognizes this and OSHA recognizes it?

A. Well, it's important from a preventive health point of view, because a disease that has been characterized and recognized is preventable. And the ways to prevent it have also been well characterized in terms of reducing or suppressing dust, wetting isolation,

ventilation, exhaust, use of respirators, substitution. All of those things have been characterized. So there are effective ways to reduce risk, and that's what the goal is of EPA and OSHA.

Q. And this reduction of risk. Are you aware of not only statements by OSHA and EPA but published peer review articles where they discuss there not being a threshold for exposure?

A. Yes. I mean the important thing to realize here is that the regulatory limits, whether they be the threshold limit values or the permissible exposure limits derived from them, are not bright line levels for safety. They were never recognized as such. For asbestos those regulatory limits were made for asbestosis, the scarring disease, not for Mesothelioma.

And in terms of the permissible limits. OSHA has to consider feasibility, as well as the recommended limits for health, and they recognize that disease occurs at the permissible exposure limits. In fact, the study that the current permissible exposure limit is based on predicts that there would be five respiratory cancers per thousand individuals. So it's not a bright line safe level. And OSHA developed an action level that's less than the permissible exposure of 50 percent of it to take action at lower levels, recognizing that there is risk at

the permissible limit.
Q. And I have a quote up there about the .1 fiber per cc level: "There still leaves a remaining significant risk." That's what you're talking about with the OSHA level; right?
A. That's correct. Yes.
Q. In fact, the reason that level was set was not because of some fine line in the sand between safe and unsafe. It was because, as OSHA said, they believed this is the practical lower limit of feasibility for measuring asbestos levels.
A. Yes. OSHA has to consider the feasibility of their permissible limits as well.
Q. And the article I had from Schall, what year was that article?
A. I believe that was out of the 1960s.
Q. And so this idea that there's no fine line between safe and dangerousness as regards to asbestos. That's been known in the literature from at least the '60s?
A. Yes. And earlier. Certainly Stokinger and others have spoke of the risk and permissible limits even in the 1950s.
Q. So it's not just OSHA and the EPA that says this stuff; it's other folks in the literature?
A. Yes.

Q. Now, the last area I want to talk to you about is fiber potency between Chrysotile and Amphibole. Is that an important distinction when we're looking at individuals that have been exposed to asbestos when you're looking at them in the clinic?

A. I would say clinically it's not an important issue. I mean, certainly, it's something that's discussed in the scientific literature. I believe it's my opinion, based on the evidence, there is a potency difference, but it's not clinically important. When physicians such as myself take an occupational history, we're not focused on getting the Amphibole history and not getting the Chrysotile history. We want the history of asbestos exposure and that's because all of the fiber types are potent in causing asbestos-related diseases, including Mesothelioma.

In my opinion, the difference in potency is in the range of about threefold difference. It's not a bright line level of potency difference. But the important fact is that Chrysotile, as well as the Amphiboles, are potent in causing Mesothelioma. Chrysotile is a known human carcinogen Group 1A. So, in the history, I'm not distinguishing, and others in my field aren't distinguishing, Amphiboles from Chrysotile. I don't think it's clinically important from in that respect,

although I'm certainly aware there's a scientific discussion of potency difference.

Q. In fact, the Helsinki criteria that we began part of our discussion with. Even though they don't distinguish between fiber types and they say that low levels of exposure to asbestos can be attributed to Mesothelioma causation, they also understood that there was this question in the literature whether Chrysotile, and there was some potency difference. Correct?

A. Well, Helsinki indicates that Amphiboles show a greater carcenogenic potency than Chrysotile and, I think, primarily related to Mesothelioma. I agree with that statement. I think it correctly reflects the epidemiological literature.

Q. And Dr. Brodkin, based upon all of the things that we've reviewed -- looking at the Helsinki criteria, looking at the Sir Bradford-Hill criteria, applying all of those to the world's literature -- do you have an opinion as to whether Chrysotile asbestos, the type of asbestos found in gaskets and packing, can cause Mesothelioma in human beings?

A. In my opinion, Chrysotile in gaskets and packing is a potent risk factor for development of Mesothelioma. And an individual exposed with sufficient dose to gaskets and packing with related Chrysotile exposure would be at

increased risk for developing Mesothelioma. It is a well documented cause of Mesothelioma.

Q. And doctor, we didn't ask you to look at all the different individuals that have filed claims in this case; correct?

A. That's true.

Q. And in regards to your opinions. Have you held all of those within a reasonable degree of medical possibility?

A. Yes.

Q. Your Honor, at this time we would offer Dr. Brodkin's CV, which is ACC-3332; his report, which is ACC-3333; his rebuttal report, which is ACC-3334; and we would offer the Power Point which is going to be ACC-3336 into evidence.

MR. SCHACHTER: The only objection I have, Your Honor, is to the Power Point on Becklake. That's a study that we've looked for and it's not in his references and shouldn't be on there and shouldn't be talked about. Otherwise --

MR. FROST: Your Honor, we'll review it. And if it's not in his materials, I'll excise it out of the Power Point that I submit to the Court.

THE COURT: All right. If it's not in there, I'm going to sustain the objection. Otherwise, I'll accept

it.

MR. FROST: With that, Your Honor, I'll pass the witness.

MR. SCHACHTER: For clarification. Those are for demonstrative purposes; right?

MR. FROST: His CV is for substantive purposes, and the rest are the way we've offered all the other materials.

THE COURT: Let's get Mr. Guy's questions and then we'll break for the day.

MR. GUY: Your Honor, this is the one time I need an hour.

(Laughter.)

THE COURT: You can have it. I won't be here.

MR. GUY: I don't need it.

THE COURT: Turn out the lights when you get done.

(Laughter.)

CROSS-EXAMINATION

BY MR. GUY:

Q. The whole issue of science and asbestos may have been beaten to death last week, and we probably need a new whip. Dr. Brodkin, I'm not going to ask you the question that I asked the debtor's experts about whether it was known in the public arena, the science, because I think that's pretty much established now that they

understood it. What I want to ask you about is, you testified as to your understanding of the status of the science concerning that issue; correct?

A. Yes, we did talk about the evolution of medical knowledge.

Q. And you testified, by reference, to numerous studies; correct?

A. Yes.

Q. Some of which have dated back decades?

A. True.

Q. Now you may not be able to answer this question. And if you can't, that's okay. Do you believe that the status of the science will change dramatically in the next five to ten years?

A. I think with asbestos-related disease, as many other areas of medicine, there will likely be further refinement of the knowledge. I don't think there will be drastic changes in terms of the evidence of causation, although there may be additional refinement. I mean, asbestos-related diseases now have been studied for many decades and, I think, been well established. So I think the new knowledge will be fine tuning or refinement of knowledge, rather than dramatic new knowledge. I mean, one doesn't have a crystal ball. But I think given the longitudinal history of asbestos studies, I don't think

there will be dramatic differences in the areas I've testified to.

Q. So you don't expect, you know, in the near future that there will be some study that would come out that would find your conclusion to be completely wrong?

MR. SCHACHTER: Objection, Your Honor. This is rank speculation.

THE COURT: I think so. We'll sustain that objection.

MR. GUY: We have all we need, Your Honor. Thank you.

THE COURT: All right. Thank you.

All right. We'll break for the night and come back at 9:30 in the morning.

(Off the record at 5:24 p.m.)

CERTIFICATE

I, Tracy Rae Dunlap, RMR, CRR, an Official Court Reporter for the United States District Court for the Western District of North Carolina, do hereby certify that I transcribed, by machine shorthand, the proceedings had in the case of IN RE: GARLOCK SEALING TECHNOLOGIES, LLC, et al, Bankruptcy Case No. 10-BK-31607, on July 30, 2013.

In witness whereof, I have hereto subscribed my name, this 31st day of July 2013.

__/S/__Tracy Rae Dunlap___
TRACY RAE DUNLAP, RMR, CRR
OFFICIAL COURT REPORTER